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=> file biosis caba caplus embase japio lifesci medline scisearch
=> s (ineffective immune response?)
L1         247 (INEFFECTIVE IMMUNE RESPONSE?)

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2          77 DUP REM L1 (170 DUPLICATES REMOVED)

=> s l2 and tuberculosis
L3           2 L2 AND TUBERCULOSIS

=> d bib ab kwic 1-
YOU HAVE REQUESTED DATA FROM 2 ANSWERS - CONTINUE? Y/(N):y

L3   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2010 ACS on STN
AN   2003:542785  CAPLUS <<LOGINID::20100316>>
DN   139:115941
TI   Dynamics of cytokine generation in patients with active pulmonary
       ***tuberculosis***
AU   Jo, Eun-Kyeong; Park, Jeong-Kyu; Dockrell, Hazel M.
CS   Department of Microbiology, College of Medicine, Chungnam National
     University, Daejeon, S. Korea
SO   Current Opinion in Infectious Diseases (2003), 16(3), 205-210
     CODEN: COIDE5; ISSN: 0951-7375
PB   Lippincott Williams & Wilkins
DT   Journal; General Review
LA   English
AB   A review.  Cytokines have been implicated in the protective immunity,
     pathophysiol., and development of   ***tuberculosis***  .  Most people who
     become infected with Mycobacterium   ***tuberculosis***   mount an
     effective protective immune response, but 5-10% develop disease.  Active
     pulmonary   ***tuberculosis***   can be considered to reflect an
       ***ineffective***     ***immune***     ***response***   against
     mycobacterial infection.  A better understanding of how cytokine prodn.
     contributes to immunity and pathol. would aid the development of new
     vaccines and therapeutic strategies.  At the time of diagnosis, prodn. of
     M.   ***tuberculosis***   or mycobacterial antigen-induced
     interferon-.gamma. by peripheral blood mononuclear cells from
       ***tuberculosis***   patients is usually depressed, compared with that of
     healthy control subjects, whereas cytokine prodn. at the site of disease
     is elevated.  In most patients, depressed interferon-.gamma. prodn. by
     peripheral blood mononuclear cells seems to be a transient response
     because it is increased in most active   ***tuberculosis***   patients
     during and following successful antituberculous therapy.  However, some
     patients remain anergic in vivo and in vitro after chemotherapy.  Among
     the cytokines contributing to protective immunity, interleukins 12 and 18,
     and tumor necrosis factor-.alpha. are important.
OSC.G   31    THERE ARE 31 CAPLUS RECORDS THAT CITE THIS RECORD (31 CITINGS)
RE.CNT  52    THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD
              ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI   Dynamics of cytokine generation in patients with active pulmonary
       ***tuberculosis***
AB   A review.  Cytokines have been implicated in the protective immunity,
     pathophysiol., and development of   ***tuberculosis***  .  Most people who
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     effective protective immune response, but 5-10% develop disease.  Active
     pulmonary   ***tuberculosis***   can be considered to reflect an
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ineffective ***immune*** ***response*** against mycobacterial infection. A better understanding of how cytokine prodn. contributes to immunity and pathol. would aid the development of new vaccines and therapeutic strategies. At the time of diagnosis, prodn. of M. ***tuberculosis*** or mycobacterial antigen-induced interferon-.gamma. by peripheral blood mononuclear cells from ***tuberculosis*** patients is usually depressed, compared with that of healthy control subjects, whereas cytokine prodn. at the site of disease is. . . interferon-.gamma. prodn. by peripheral blood mononuclear cells seems to be a transient response because it is increased in most active ***tuberculosis*** patients during and following successful antituberculous therapy. However, some patients remain anergic in vivo and in vitro after chemotherapy. Among. . .

ST review cytokine pulmonary ***tuberculosis***

IT Immune tolerance
(anergy; cytokine generation in patients with active pulmonary ***tuberculosis*** in relation to T cell anergy)

IT Human
Mycobacterium ***tuberculosis***
Tuberculosis
(cytokine generation in patients with active pulmonary ***tuberculosis***)

IT Cytokines
Interleukin 12
Interleukin 18
Tumor necrosis factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(cytokine generation in patients with active pulmonary ***tuberculosis***)

IT T cell (lymphocyte)
(cytokine generation in patients with active pulmonary ***tuberculosis*** in relation to T cell anergy)

IT Interferons
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(.gamma.; cytokine generation in patients with active pulmonary ***tuberculosis***)

L3 ANSWER 2 OF 2 LIFESCI COPYRIGHT 2010 CSA on STN
AN 96:75735 LIFESCI <<LOGINID::20100316>>
TI IL12 and the human immune response to mycobacteria
AU Modlin, R.L.; Barnes, P.F.
CS UCLA Sch. Med., 52-121 CHS, 10833 LeConte Ave., Los Angeles, CA 9009-1750, USA
SO RES. IMMUNOL., (1995) vol. 146, no. 7-8, pp. 526-531.
ISSN: 0923-2494.
DT Journal
TC General Review
FS F; J
LA English
AB Human infection with Mycobacterium ***tuberculosis*** results in a broad spectrum of outcomes ranging from asymptomatic infection to widespread and rapidly fatal disease. It is generally believed that this spectrum of clinical manifestations reflects a complex interaction between M. ***tuberculosis*** and human immune defences. Most persons who become infected with M. ***tuberculosis*** mount a protective immune response and remain clinically well, the only evidence of infection being development of a positive tuberculin skin test. Only a minority of

infected persons develop disease, the most serious form being miliary ***tuberculosis*** , characterized by haematogenous dissemination of organisms throughout the body, and severe disease that is almost invariably fatal if untreated. These manifestations reflect an ***ineffective*** ***immune*** ***response*** , manifested by a high frequency of negative tuberculin skin tests and failure of T cells to proliferate in response to M. ***tuberculosis*** antigens. Between the extremes of effective and ineffective responses to M. ***tuberculosis*** , two common manifestations of ***tuberculosis*** reflect intermediate immune responses. Tuberculous pleuritis results when a small focus of organisms ruptures into the pleural space, triggering an exudative pleural effusion from a vigorous delayed-type hypersensitivity response. Patients with pleuritis mount a relatively resistant immune response to infection, reflected by resolution of pleuritis without therapy in most cases, and a high frequency of positive tuberculin skin tests. In contrast, patients with advanced pulmonary ***tuberculosis*** have an moderately ***ineffective*** ***immune*** ***response*** . They often develop progressive and life-threatening disease, and 20-30 % of patients have negative tuberculin skin tests.

AB Human infection with Mycobacterium ***tuberculosis*** results in a broad spectrum of outcomes ranging from asymptomatic infection to widespread and rapidly fatal disease. It is generally believed that this spectrum of clinical manifestations reflects a complex interaction between M. ***tuberculosis*** and human immune defences. Most persons who become infected with M. ***tuberculosis*** mount a protective immune response and remain clinically well, the only evidence of infection being development of a positive tuberculin skin test. Only a minority of infected persons develop disease, the most serious form being miliary ***tuberculosis*** , characterized by haematogenous dissemination of organisms throughout the body, and severe disease that is almost invariably fatal if untreated. These manifestations reflect an ***ineffective*** ***immune*** ***response*** , manifested by a high frequency of negative tuberculin skin tests and failure of T cells to proliferate in response to M. ***tuberculosis*** antigens. Between the extremes of effective and ineffective responses to M. ***tuberculosis*** , two common manifestations of ***tuberculosis*** reflect intermediate immune responses. Tuberculous pleuritis results when a small focus of organisms ruptures into the pleural space, triggering an. . . without therapy in most cases, and a high frequency of positive tuberculin skin tests. In contrast, patients with advanced pulmonary ***tuberculosis*** have an moderately ***ineffective*** ***immune*** ***response*** . They often develop progressive and life-threatening disease, and 20-30 % of patients have negative tuberculin skin tests.

UT interleukin 12; Mycobacterium ***tuberculosis*** ; immune response; reviews

=> s l2 and HIV
L4 5 L2 AND HIV

=> d bib ab kwic 1-
YOU HAVE REQUESTED DATA FROM 5 ANSWERS - CONTINUE? Y/(N):y

L4 ANSWER 1 OF 5 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2002:461568 BIOSIS <<LOGINID::20100316>>
DN PREV200200461568
TI A Tat subunit vaccine confers protective immunity against the

immune-modulating activity of the human immunodeficiency virus type-1 Tat protein in mice.

AU Agwale, S. M.; Shata, M. T.; Reitz, M. S., Jr.; Kalyanaraman, V. S.; Gallo, R. C.; Popovic, M.; Hone, D. M. [Reprint author]

CS Division of Vaccine Research, University of Maryland Biotechnology Institute, Baltimore, MD, 21202, USA
hone@umbi.umd.edu

SO Proceedings of the National Academy of Sciences of the United States of America, (July 23, 2002) Vol. 99, No. 15, pp. 10037-10041. print.
CODEN: PNASA6. ISSN: 0027-8424.

DT Article

LA English

ED Entered STN: 28 Aug 2002
Last Updated on STN: 28 Aug 2002

AB The rational design of new therapies against ***HIV*** -1 necessitates an improved understanding of the mechanisms underlying the production of ***ineffective*** ***immune*** ***responses*** to ***HIV*** -1 in most infected individuals. This report shows that the CD8+ T cell responses to gp120 were greatly diminished in mice vaccinated with a bicistronic gp120-Tat DNA vaccine, compared with those induced by a DNA vaccine encoding gp120 alone. The CD8+ T cell responses induced by the latter included strong gp120-specific IFN-gamma secretion and protective antiviral immunity against challenge by a vaccinia-env pseudotype. The degree to which Tat influenced CD8+ T cell responses depended on the bioactivity of Tat. Thus, a bicistronic DNA vaccine that expresses gp120 and a truncated Tat defective for LTR activation elicited strong IFN-gamma-secreting CD8+ T cell responses to gp120 but conferred only marginal protection against the vaccinia-env challenge. The effect of Tat was completely blocked, however, by immunization with inactivated Tat protein before vaccination with the bicistronic gp120-Tat DNA vaccine.

AB The rational design of new therapies against ***HIV*** -1 necessitates an improved understanding of the mechanisms underlying the production of ***ineffective*** ***immune*** ***responses*** to ***HIV*** -1 in most infected individuals. This report shows that the CD8+ T cell responses to gp120 were greatly diminished in mice. . .

L4 ANSWER 2 OF 5 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

AN 1997:393692 BIOSIS <<LOGINID::20100316>>

DN PREV199799692895

TI The viral envelope in the evolution of ***HIV*** : A hypothetical approach to inducing an effective immune response to the virus.

AU Ngu, V. A.

CS Cancer Res. Lab., Fac. Med. Biomed. Sci., B.P. 1364, Yaounde, Cameroon

SO Medical Hypotheses, (1997) Vol. 48, No. 6, pp. 517-521.
CODEN: MEHYDY. ISSN: 0306-9877.

DT Article

LA English

ED Entered STN: 10 Sep 1997
Last Updated on STN: 10 Sep 1997

AB The human immunodeficiency virus (***HIV***) is 'perceived' by the host immune system as partly-self because of the presence of host cell wall membrane on the viral envelope. This perception leads to an ***ineffective*** ***immune*** ***response*** to the virus. It is proposed that only viral core antigens without the envelope will be perceived as non-self by the host immune system and can provoke an effective immune response. In normal uninfected persons, core antigens could therefore serve as a vaccine. In ***HIV*** infected persons,

uncommitted immunocytes from the peripheral leucocytes freed from antibodies will in vitro process autologous viral core antigens as non-self antigens and lead to an effective immune response against the ***HIV*** when reinjected into the patient. The use of autologous viral core antigens provides, at the same time, a means for testing viral core antigens as possible vaccines without any risk to a third person. This immunotherapy of the ***HIV*** , when confirmed, will support core antigens as possible vaccines and could also be applied to the large group of retroviral and other enveloped viruses that cause chronic infections and malignant tumours in man and animals, with considerable benefits to human and animal health.

TI The viral envelope in the evolution of ***HIV*** : A hypothetical approach to inducing an effective immune response to the virus.

AB The human immunodeficiency virus (***HIV***) is 'perceived' by the host immune system as partly-self because of the presence of host cell wall membrane on the viral envelope. This perception leads to an ***ineffective*** ***immune*** ***response*** to the virus. It is proposed that only viral core antigens without the envelope will be perceived as non-self by. . . and can provoke an effective immune response. In normal uninfected persons, core antigens could therefore serve as a vaccine. In ***HIV*** infected persons, uncommitted immunocytes from the peripheral leucocytes freed from antibodies will in vitro process autologous viral core antigens as non-self antigens and lead to an effective immune response against the ***HIV*** when reinjected into the patient. The use of autologous viral core antigens provides, at the same time, a means for testing viral core antigens as possible vaccines without any risk to a third person. This immunotherapy of the ***HIV*** , when confirmed, will support core antigens as possible vaccines and could also be applied to the large group of retroviral. . .

IT Miscellaneous Descriptors
AUTOLOGOUS VIRAL CORE ANTIGEN; ***HIV*** INFECTION; HOST; HOST IMMUNE RESPONSE; HUMAN IMMUNODEFICIENCY VIRUS INFECTION; IMMUNE SYSTEM; INFECTION; PATHOGEN; PATHOVAR-EVOLUTION; POTENTIAL VACCINE; VIRAL DISEASE; VIRAL ENVELOPE

ORGN . . .
Primates, Vertebrates

ORGN Classifier
Retroviridae 03305

Super Taxa
DNA and RNA Reverse Transcribing Viruses; Viruses; Microorganisms

Organism Name
human immunodeficiency virus
HIV

Taxa Notes
DNA and RNA Reverse Transcribing Viruses, Microorganisms, Viruses

L4 ANSWER 3 OF 5 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

AN 1996:562637 BIOSIS <<LOGINID::20100316>>

DN PREV199799291993

TI Molecular analysis of mixed infection with hepatitis C virus and human immunodeficiency virus in a patient infected simultaneously.

AU Mazza, Cinzia; Puoti, Massimo; Ravaggi, Antonella; Castelnuovo, Filippo; Albertini, Alberto; Cariani, Elisabetta [Reprint author]

CS III Lab. Analisi, Spedali Civili, p.le Spedali Civili 1, 25123 Brescia, Italy

SO Journal of Medical Virology, (1996) Vol. 50, No. 3, pp. 276-282.

CODEN: JMVIDB. ISSN: 0146-6615.
DT Article
LA English
ED Entered STN: 23 Dec 1996
Last Updated on STN: 23 Dec 1996
AB A case of simultaneous infection with ***HIV*** and HCV characterized by a rapidly progressive clinical course was studied retrospectively over 3.5 years. Molecular analysis indicated interference between ***HIV*** and HCV and between HCV subtypes 1a and 1b. An ***ineffective*** ***immune*** ***response*** was suggested by the persistence and sequence conservation of the HCV HVR1 variants isolated during the follow-up.
AB A case of simultaneous infection with ***HIV*** and HCV characterized by a rapidly progressive clinical course was studied retrospectively over 3.5 years. Molecular analysis indicated interference between ***HIV*** and HCV and between HCV subtypes 1a and 1b. An ***ineffective*** ***immune*** ***response*** was suggested by the persistence and sequence conservation of the HCV HVR1 variants isolated during the follow-up.

L4 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2005:395589 CAPLUS <<LOGINID::20100316>>
DN 142:423905
TI Method of therapy of disease based on immune cycling and presence of regulator cells
IN Ashdown, Martin Leonard
PA Immunaid Pty. Ltd., Australia
SO PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005040816	A1	20050506	WO 2004-AU1456	20041022
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2004283322	A1	20050506	AU 2004-283322	20041022
	CA 2543490	A1	20050506	CA 2004-2543490	20041022
	EP 1692516	A1	20060823	EP 2004-761461	20041022
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				
	BR 2004015533	A	20061226	BR 2004-15533	20041022
	CN 1898569	A	20070117	CN 2004-80038999	20041022
	JP 2007509078	T	20070412	JP 2006-535913	20041022
	MX 2006004522	A	20061110	MX 2006-4522	20060424
	US 20070202119	A1	20070830	US 2007-576981	20070302
PRAI	AU 2003-905858	A	20031024		

WO 2004-AU1456 W 20041022

AB The present inventor has surprisingly found that the immune system is cycling during disease states characterized by the presence of regulator cells. While not wishing to be limited by theory, it appears that effector cell expansion against a target antigen is followed by the expansion of regulator cells directed against the effectors. Upon control of the effector cells by the regulator cells the nos. and/or activity of both types of cells decrease, which in turn is followed by the same cycle due to the continuous presence or incomplete removal of antigen which results in an oscillating persistent, but ***ineffective*** , ***immune*** ***response*** against the, for example, tumor or virus. The present invention provides a method of treating a disease characterized by the prodn. of regulator cells, the method comprising; (i) monitoring a patient suffering from the disease for at least one of: (a) no. and/or activity of regulator cells, (b) no. and/or activity of effector cells, (c) a mol. assocd. with the disease, and/or (d) an immune system marker, and (ii) exposing the patient to an agent to treat the disease, wherein the timing of administration of the agent is selected such that the activity of the effector cells is not significantly reduced. Based on these observations, the present invention provides methods for treating diseases such as cancer and a ***HIV*** infection.

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB . . . the same cycle due to the continuous presence or incomplete removal of antigen which results in an oscillating persistent, but ***ineffective*** , ***immune*** ***response*** against the, for example, tumor or virus. The present invention provides a method of treating a disease characterized by the. . . not significantly reduced. Based on these observations, the present invention provides methods for treating diseases such as cancer and a ***HIV*** infection.

ST disease treatment immune cycling regulator cell; cancer treatment immune cycling regulator cell; ***HIV*** infection immune cycling regulator cell

L4 ANSWER 5 OF 5 EMBASE

AN 1997192505 EMBASE <<LOGINID::20100316>>

TI [Cytokines and ***HIV*** infection. Pathophysiology, diagnosis and therapeutic strategies].
Cytokines et infection par le virus de l'immunodeficience humaine (VIH-1): Implications physiopathologiques et consequences diagnostiques et therapeutiques.

AU Guenounou, M. (correspondence)
AU Guenounou, M. (correspondence)
CS Lab. Immunologie/Biologie Cytokines, Centre des Biomolecules, Universite de Reims, 51, Rue Cognacq-Jay, 51096 Reims Cedex 01, France.

SO Immuno-Analyse et Biologie Specialisee, (May 1997) Vol. 12, No. 2, pp. 65-69.
Refs: 50
ISSN: 0923-2532 CODEN: IBSPEW
CY France
DT Journal; General Review; (Review)
FS 026 Immunology, Serology and Transplantation
004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
005 General Pathology and Pathological Anatomy
LA French
SL French; English

ED Entered STN: 7 Aug 1997
Last Updated on STN: 7 Aug 1997
AB The ***HIV*** infection paradox is that it induces a hyper-activation of the immune system resulting in an ***ineffective*** ***immune*** ***response*** . In this context, the cytokine network, which plays a key role in the regulation of cellular interactions within the immune system, is highly challenged. An imbalance in the cytokine profile resulting from cytokine disruption by ***HIV*** can be associated to disease progression, and cytokines may be involved in viral replication. Cytokines, cytokine inhibitors, and several cell derived anti-viral factors are proposed in immune based therapeutic strategies. In this review we attempt to analyse the involvement of cytokines in ***HIV*** -induced immune hyper-activation, the consequences of ***HIV*** infection on the cytokine imbalanced and T-cell defect and the appearance of ***HIV*** related haematological disorders and oncogenesis. Potential use of cytokine detection for prognostic purposes and insights in immune-based therapeutic strategies are discussed.
TI [Cytokines and ***HIV*** infection. Pathophysiology, diagnosis and therapeutic strategies].
Cytokines et infection par le virus de l'immunodeficience humaine (VIH-1): Implications physiopathologiques et consequences. . .
AB The ***HIV*** infection paradox is that it induces a hyper-activation of the immune system resulting in an ***ineffective*** ***immune*** ***response*** . In this context, the cytokine network, which plays a key role in the regulation of cellular interactions within the immune system, is highly challenged. An imbalance in the cytokine profile resulting from cytokine disruption by ***HIV*** can be associated to disease progression, and cytokines may be involved in viral replication. Cytokines, cytokine inhibitors, and several cell. . . factors are proposed in immune based therapeutic strategies. In this review we attempt to analyse the involvement of cytokines in ***HIV*** -induced immune hyper-activation, the consequences of ***HIV*** infection on the cytokine imbalanced and T-cell defect and the appearance of ***HIV*** related haematological disorders and oncogenesis. Potential use of cytokine detection for prognostic purposes and insights in immune-based therapeutic strategies are. . .

=> s l2 and (ineffective/ti)
L5 4 L2 AND (INEFFECTIVE/TI)

=> d 1-
YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L5 ANSWER 1 OF 4 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2000:349508 BIOSIS <<LOGINID::20100316>>
DN PREV200000349508
TI ***Ineffective*** cellular immune response associated with T-cell apoptosis in susceptible Mycobacterium bovis BCG-infected mice.
AU Kremer, Laurent; Estaquier, Jerome [Reprint author]; Wolowczuk, Isabelle; Biet, Franck; Ameisen, Jean-Claude; Locht, Camille
CS INSERM E9922, Groupe Hospitalier Bichat-Claude Bernard, 16 Rue Henri Huchard, 75018, Paris, France
SO Infection and Immunity, (July, 2000) Vol. 68, No. 7, pp. 4264-4273. print.
CODEN: INFIBR. ISSN: 0019-9567.
DT Article
LA English

ED Entered STN: 16 Aug 2000
    Last Updated on STN: 7 Jan 2002

L5 ANSWER 2 OF 4 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 1979:57760 BIOSIS <<LOGINID::20100316>>
DN PREV197916057760; BR16:57760
TI EFFECTIVE AND ***INEFFECTIVE*** ***IMMUNE*** ***RESPONSES***
    TO PARASITES EVIDENCE FROM EXPERIMENTAL MODELS.
AU PLAYFAIR J H L
SO (1978) pp. 37-64. ARBER, W. ET AL. (ED.). CURRENT TOPICS IN MICROBIOLOGY
    AND IMMUNOLOGY, VOL. 80. III+169P. ILLUS. SPRINGER-VERLAG: NEW YORK, N.Y.,
    USA; BERLIN, WEST GERMANY. ISBN 0-387-08781-8; ISBN 3-540-08781-8.
DT Book
FS BR
LA Unavailable
ED Entered STN: 28 Apr 1986
    Last Updated on STN: 28 Apr 1986

L5 ANSWER 3 OF 4 CABA COPYRIGHT 2010 CABI on STN
AN 79:114717 CABA <<LOGINID::20100316>>
DN 19792238076
TI Effective and ***ineffective*** ***immune*** ***responses***
    to parasites: evidence from experimental models
AU Playfair, J. H. L.
CS Dep. Immunol., Middlesex Med. Sch., London W1P 9PS, UK.
SO Current Topics in Microbiology and Immunology, (1978) Vol. 80, pp. 37-64.
    Refs. pp.60-64.
    ISSN: 0070-217X
DT Journal
LA English
ED Entered STN: 1 Nov 1994
    Last Updated on STN: 1 Nov 1994

L5 ANSWER 4 OF 4 CABA COPYRIGHT 2010 CABI on STN
AN 79:54576 CABA <<LOGINID::20100316>>
DN 19790856234
TI Effective and ***ineffective*** ***immune*** ***responses***
    to parasites: evidence from experimental models
AU Playfair, J. H. L.; : Arber, W. (et al.) [EDITOR]
CS Dep. of Immunology, Arthur Stanley House, The Middlesex Med. School, 40-50
    Tottenham Street, London W1P 9PG, UK.
SO Current topics in microbiology and immunology. Volume 80, (1978) pp.
    37-64.
    Publisher: Springer-Verlag. Berlin
CY Germany, Federal Republic of
DT Miscellaneous
LA English
ED Entered STN: 1 Nov 1994
    Last Updated on STN: 1 Nov 1994

=> s l2 and ineffective/ab
'AB' IS NOT A VALID FIELD CODE
'AB' IS NOT A VALID FIELD CODE
'AB' IS NOT A VALID FIELD CODE
L6   68 L2 AND INEFFECTIVE/AB

=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 68 ANSWERS - CONTINUE? Y/(N):y

L6 ANSWER 1 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2010:125809 BIOSIS <<LOGINID::20100316>>
DN PREV201000125809
TI Recent Insights in Primary Immunodeficiency Diseases: The Role of T-Lymphocytes and Innate Immunity.
AU Pandolfi, Franco [Reprint Author]; Cianci, Rossella; Cammarota, Giovanni; Pagliari, Danilo; Landolfi, Raffaele; Conti, Pio; Theoharides, Theoharis C.
CS Univ Cattolica Sacro Cuore, Inst Internal Med, Largo F Vito 1, I-00186 Rome, Italy
pandolfi@rm.unicatt.it; pconti@unich.it
SO Annals of Clinical and Laboratory Science, (WIN 2010) Vol. 40, No. 1, pp. 3-9.
CODEN: ACLSCP. ISSN: 0091-7370. E-ISSN: 1550-8080.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 3 Mar 2010
Last Updated on STN: 3 Mar 2010
AB In recent years, the field of primary immunodeficienciency diseases (PID) has experienced remarkable progress with the identification of a number of new genes associated with specific diseases. Yet the diagnosis of PID remains difficult. In fact, this field requires continuous updating because once a novel molecule related to the immune function is discovered, the corresponding PID will soon be described. Since comprehensive reviews on the classification of PID are available, we concentrate here on reviewing some controversial and new issues, mainly those related to the role of T-cells and innate immunity. We will consider common variable immunodeficiency as an example of a PID where several immune pathways are impaired. We will also discuss the restricted usage of the T-cell receptor repertoire in PID. Innate immunity and Toll-like receptors (TLR) are new major players in this field. We will therefore discuss the association of TLR with the function of Bruton tyrosine kinase (Btk) that is essential in the development of B-cells and in the pathogenesis of X-linked agammaglobulinemia. Finally, we will discuss the role of mast-cells. These cells were once thought to be relevant almost exclusively to the pathogenesis of allergy. Now we know that mast cells are involved in initiating the adaptive response and may contribute to ***ineffective*** ***immune*** ***responses*** .

L6 ANSWER 2 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2009:380174 BIOSIS <<LOGINID::20100316>>
DN PREV200900381277
TI Prostate cancer lesions are surrounded by FOXP3(+), PD-1(+) and B7-H1(+) lymphocyte clusters.
AU Ebelt, Kathleen [Reprint Author]; Babaryka, Gregor; Frankenberger, Bernhard; Stief, Christian G.; Eisenmenger, Wolfgang; Kirchner, Thomas; Schendel, Dolores J.; Noessner, Elfriede
CS German Res Ctr Environm Hlth GmbH, Helmholtz Ctr Munich, Inst Mol Immunol, Marchioninistr 25, D-81377 Munich, Germany
ebelt.k@web.de
SO European Journal of Cancer, (JUN 2009) Vol. 45, No. 9, pp. 1664-1672.
CODEN: EJCAEL. ISSN: 0959-8049.
DT Article

LA English
ED Entered STN: 24 Jun 2009
Last Updated on STN: 18 Nov 2009
AB The immune response against prostate cancer seems to be inefficient although tumour cells show an over-expression of tumour-associated antigens suggesting that regulatory networks inhibit immune cell function locally. To address this proposition, lymphocytes within prostate cancer-inflicted tissue were analysed for the expression of markers associated with negative regulatory function and exhaustion.Prostate cancer, benign prostatic hyperplasia and healthy prostate tissues were investigated by immunohistology for CD25, FOXP3, PD-1 and B7-H1.We had previously documented that prostate cancer islets are surrounded by clustered accumulations of CD3(+) lymphocytes, which lack perforin and interferon-gamma (IFN gamma) expression, thus are apparently quiescent. Here, we report that these clusters contain numerous CD25(+) and FOXP3(+) cells. These markers are associated with regulatory T cells, and their presence in lymphocyte clusters near prostate cancer regions indicates an environment with negative impact on immune response against cancer cells. Consistent with this hypothesis, cells expressing PD-1 and its ligand B7-H1, which are markers associated with exhaustion of lymphocyte function, were also detected in the lymphocyte clusters.Expression of molecules associated with inhibition and exhaustion of lymphocytes may reflect events contributing to ***ineffective*** ***immune*** ***responses*** against cancer cells. (C) 2009 Elsevier Ltd. All rights
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L6 ANSWER 3 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2009:362774 BIOSIS <<LOGINID::20100316>>
DN PREV200900363877
TI Necator americanus Infection: A Possible Cause of Altered Dendritic Cell Differentiation and Eosinophil Profile in Chronically Infected Individuals.
AU Fujiwara, Ricardo T. [Reprint Author]; Cancado, Guilherme G. L.; Freitas, Paula A.; Santiago, Helton C.; Massara, Cristiano Lara; Carvalho, Omar dos Santos; Correa-Oliveira, Rodrigo; Geiger, Stefan M.; Bethony, Jeffrey
CS Fundacao Oswaldo Cruz, Inst Rene Rachou, Cellular and Mol Immunol Lab, Belo Horizonte, MG, Brazil
jeff@cpqrr.fiocruz.br
SO PLoS Neglected Tropical Diseases, (MAR 2009) Vol. 3, No. 3, pp. Article No.: e399. http://www.plosntds.org/static/information.action.
ISSN: 1935-2735.
DT Article
LA English
ED Entered STN: 11 Jun 2009
Last Updated on STN: 11 Jun 2009
AB Background: Hookworms survive for several years (5 to 7 years) in the host lumen, inducing a robust but largely ***ineffective*** ***immune*** ***response*** . Among the most striking aspects of the immune response to hookworm (as with many other helminths) is the ablation of parasite-specific T cell proliferative response (hyporesponsiveness). While the role of the adaptive immune response in human helminth infection has been well investigated, the role of the innate immune responses (e. g., dendritic cells and eosinophils) has received less attention and remains to be clearly elucidated.Methodology/Principal Findings: We report on the differentiation/maturation of host dendritic cells in vitro and the eosinophil activation/function associated with human hookworm infection.

Mature DCs (mDCs) from Necator americanus (Necator)-infected individuals showed an impaired differentiation process compared to the mDCs of non-infected individuals, as evidenced by the differential expression of CD11c and CD14. These same hookworm-infected individuals also presented significantly down-regulated expression of CD86, CD1a, HLA-ABC, and HLA-DR. The lower expression of costimulatory and antigen presentation molecules by hookworm-infected-derived mDCs was further evidenced by their reduced ability to induce cell proliferation. We also showed that this alternative DC differentiation is partially induced by excreted-secreted hookworm products. Conversely, eosinophils from the same individuals showed a highly activated status, with an upregulation of major cell surface markers. Antigen-pulsed eosinophils from N. americanus-infected individuals induced significant cell proliferation of autologous PBMCs, when compared to non-infected individuals.Conclusion: Chronic N. americanus infection alters the host's innate immune response, resulting in a possible modulation of the maturation process of DCs, a functional change that may diminish their ability for antigen presentation and thus contribute to the ablation of the parasite-specific T cell proliferative response. Interestingly, a concomitant upregulation of the major cell surface markers of eosinophils was observed in hookworm-infected individuals, indicative of antigen-specific immune responses, especially antigen presentation. We showed that in addition to the postulated role of the eosinophils as effector cells against helminth infection, activated cells may also be recruited to sites of inflammation and contribute to the immune response acting as antigen presenting cells.

L6 ANSWER 4 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2008:154938 BIOSIS <<LOGINID::20100316>>
DN PREV200800146617
TI TLR7 stimulation augments T effector-mediated rejection, of skin expressing neo-self antigen in keratinocytes.
AU Zhong, Jie; Hadis, Usriansyah; De Kluyver, Rachel; Leggatt, Graham R.; Fernando, Germain J. P.; Frazer, Ian H. [Reprint Author]
CS Univ Queensland, Princess Alexandra Hosp, Diamantina Inst Canc Immunol and Metab Med, 4th Level,Res Extens,Bldg 1, Woolloongabba, Qld 4102, Australia i.frazer@uq.edu.au
SO European Journal of Immunology, (JAN 2008) Vol. 38, No. 1, pp. 73-81. CODEN: EJIMAF. ISSN: 0014-2980.
DT Article
LA English
ED Entered STN: 27 Feb 2008
Last Updated on STN: 27 Feb 2008
AB Immunotherapy generally fails to induce tumour regression in spontaneously arising tumours. Failure is attributed to both tumour-related factors and an ***ineffective*** ***immune*** ***response*** . As a model of tumour immunotherapy, without the confounding effects of potential tumour-determined mechanisms of immune evasion, we studied the requirements for rejection of skin grafts expressing a neo-self antigen in somatic cells and not in antigen-presenting cells. When antigen expression was restricted to somatic cells, both CD4(+) and CD8(+) effector cells were required for graft rejection. Although freshly placed grafts were spontaneously rejected, healed grafts established under the cover of T cell depletion were not rejected even after T cell numbers recovered to a level where freshly placed grafts on the same animal were rejected, suggesting that healed skin grafts expressing a neo-self antigen only in somatic cells could not be rejected by primed recipients with functional effector T cells. Local TLR7 ligation induced inflammatory

responses and rejection of healed grafts exposed to the TLR agonist but did not induce rejection of untreated healed grafts on the same animal. Thus, local pro-inflammatory signalling via TLR7 can promote effector T cell function against skin cells displaying their nominal antigen.

L6 ANSWER 5 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2008:21380 BIOSIS <<LOGINID::20100316>>
DN PREV200800025142
TI Hodgkin's lymphoma associated T-cells exhibit a transcription factor profile consistent with distinct lymphoid compartments.
AU Atayar, Cigdem; van den Berg, Anke; Blokzijl, Tjasso; Boot, Marcel; Gascoyne, Randy D.; Visser, Lydia; Poppema, Sibrand [Reprint Author]
CS Univ Groningen, Med Ctr, Dept Pathol and Lab Med, Hanzepl 1,POB 30-001, NL-9700 RB Groningen, Netherlands
s.poppema@rvb.umcg.nl
SO Journal of Clinical Pathology (London), (OCT 2007) Vol. 60, No. 10, pp. 1092-1097.
CODEN: JCPAAK. ISSN: 0021-9746. E-ISSN: 1472-4146.
DT Article
LA English
ED Entered STN: 19 Dec 2007
Last Updated on STN: 19 Dec 2007
AB Background: Hodgkin's lymphoma (HL) is characterised by an ***ineffective*** ***immune*** ***response*** that is predominantly mediated by CD4(+) T-cells.Aims: To analyse the expression of the key regulatory T-cell transcription factors (TFs) in the T-cells of HL involved tissues in order to assess the nature of the T-H immune response in HL.Methods and results: By immunohistochemistry, GATA3 was strongly and T-bet exclusively expressed in a subset of interfollicular lymphocytes in the reactive lymphoid tissues. In classical HL (CHL), which is generally located in the interfollicular zones, a predominance of T-bet(+) T-cells and lesser amounts of GATA3(+) and cMaf(+) T-cells was found, concordant with the pattern of the normal interfollicular compartment. In reactive lymphoid tissues, c-Maf was observed mostly in T-lymphocytes within the germinal centres (GCs). Nodular lymphocyte predominance type of Hodgkin's lymphoma (NLPHL) and progressively transformed germinal centres cases, showed a majority of c-Maf(+) T-cells, consistent with the pattern in normal GCs. NLPHL cases uniformly showed c-Maf(+)/CD57(+) T-cell rosettes around the neoplastic cells; these rosettes were absent in "paragranuloma-type'' T-cell/histiocyte rich B-cell lymphoma.Conclusions: T-cell TF expression profiles of the reactive T-cells in both subtypes of HL are in accordance with the expression profile observed in the distinct lymphoid compartments.

L6 ANSWER 6 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2007:565814 BIOSIS <<LOGINID::20100316>>
DN PREV200700559006
TI Gross and microscopic findings and investigation of the aetiopathogenesis of mycobacteriosis in a captive population of white-winged ducks (Cairina scutulata).
AU Saggese, Miguel D. [Reprint Author]; Riggs, Gary; Tizard, Ian; Bratton, Gerald; Taylor, Robert; Phalen, David N.
CS Western Univ Hlth Sci, Coll Vet Med, 309 E 2nd St, Pomona, CA 91766 USA
msaggese@westernu.edu
SO Avian Pathology, (2007) Vol. 36, No. 5, pp. 415.
CODEN: AVPADN. ISSN: 0307-9457.
DT Article

LA English
ED Entered STN: 31 Oct 2007
Last Updated on STN: 31 Oct 2007
AB The white-winged duck (Cairina scutulata) is critically endangered. Breeding collections of this duck are established in the United Kingdom and the USA. Infection with Mycobacterium avium avium serotype 1 is a major cause of mortality in the UK collection. In this study, the aetiopathogenesis of deaths occurring in the US collection was studied. All ducks (n = 21) that died over a 21-month period were examined. Mycobacteriosis was diagnosed in 20 ducks, killing 19 of them. Multifocal to diffuse granulomatous lesions, often with abundant intralesional organisms, were seen in all 20 ducks. Unusual manifestations of this disease were the extensive involvement of the respiratory system and the absence of multinucleated giant cells. Sequence analysis showed that the ducks were infected with a sequevar of M. a. avium that contains serotypes 2, 3, 4, and 9. Given that the long-term ingestion of metals affects immune function, we measured an array of such elements in the liver of six ducks. Concentrations were undetectable or low. The disseminated nature of the disease, high concentration of mycobacteria and absence of multinucleated giant cells within lesions suggest that these ducks were unable to effectively kill the mycobacteria and point to a possible defect or inhibition in cell mediated immunity. Taken together with previously reported UK data, these results suggest that captive white-winged ducks are highly susceptible to at least two sequevars of M. a. avium and that mycobacteriosis is a major threat to ex situ breeding. We hypothesize that the minimal heterozygosis previously shown in these ducks could be contributing to an apparently ***ineffective*** ***immune*** ***response*** .

L6 ANSWER 7 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2007:362732 BIOSIS <<LOGINID::20100316>>
DN PREV200700356097
TI Serum copper concentration in adults with acute, uncomplicated Falciparum malaria infection.
AU Garba, I. H. [Reprint Author]; Ubom, G. A.; Ejiogu, N. B.
CS Abubakar Tafawa Balewa Univ, Sch Sci, Ind Chem Programme, PMB 0248, Bauchi, Nigeria
SO Biological Trace Element Research, (NOV 2006) Vol. 113, No. 2, pp. 125-130.
CODEN: BTERDG. ISSN: 0163-4984.
DT Article
LA English
ED Entered STN: 20 Jun 2007
Last Updated on STN: 20 Jun 2007
AB Serum copper concentration was measured in 80 adult patients (40 males and females each; age range: 18-40 yr) presenting with acute, uncomplicated falciparum malaria infection and a control group of 20 age-matched, healthy individuals. The mean serum copper concentration was 109.0 +/- 40.0 mu g/dL in healthy individuals. Both male and female patients were found to have a significantly decreased serum copper concentration ($p < 0.05$). In the male patients, the mean serum copper concentration decreased by 33.95%, whereas it dropped by 38.53% in their female counteraparts. A compromised enzymatic antioxidant defense capability, particularly superoxide dismutase (SOD) activity, has been reported in patients with falciparum malaria infection. Because SOD activity is dependent on copper, the ***ineffective*** SOD activity can be related to the decrease in the concentration of copper during the infection. Low

serum copper can also contribute to the ***ineffective*** ***immune*** ***response*** of the host to the antigenic challenge of the falciparum parasite because copper is also important for normal immune function.

L6 ANSWER 8 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2007:42230 BIOSIS <<LOGINID::20100316>>
DN PREV200700039134
TI Changes in hepatic immunoregulatory cytokines in patients with metastatic colorectal carcinoma: Implications for hepatic anti-tumour immunity.
AU Kelly, Anna M.; Golden-Mason, Lucy; Traynor, Oscar; Geoghegan, Justin; McEntee, Gerry; Hegarty, John E.; O'Farrelly, Cliona [Reprint Author]
CS St Vincents Univ Hosp, Educ and Res Ctr, Dublin 4, Ireland
annam_kelly@yahoo.com; lucy.golden@UCHSC.edu; mr.oscar.traynor@svcpc.ie; j.geoghegan@svcpc.ie; gerrymce@eircom.net; jhegarty@svcpc.ie; cliona.ofarrelly@ucd.ie
SO Cytokine, (AUG 2006) Vol. 35, No. 3-4, pp. 171-179.
CODEN: CYTIE9. ISSN: 1043-4666.
DT Article
LA English
ED Entered STN: 3 Jan 2007
Last Updated on STN: 3 Jan 2007
AB The hepatic immunological environment, dominated by NK and NKR+ T cells, seems specialised to respond to malignant challenge. ***Ineffective*** ***immune*** ***responses*** to malignancy are likely determined by factors including alterations in the local cytokine profile. This study examines the cytokine milieu of normal and tumour-bearing liver, quantifying pro-/anti-inflammatory cytokines using modified ELISAs and real-time quantitative PCR. Cytokine protein was localised using immunohistochemistry. We demonstrate an active cytokine environment in normal liver, with high levels of inflammatory and regulatory cytokines. Inflammatory IFN-gamma was increased in tumour-bearing liver ($p < 0.0001$). However, a much greater increase in anti-inflammatory IL-10, produced by non-parenchymal cells ($P < 0.0005$), resulted in a reduced IFN-gamma:IL-10 ratio in tumour-bearing liver ($p < 0.02$). In contrast, immunosuppressive TGF-beta and IL-13 were significantly downregulated ($p < 0.02$). Furthermore, IL-2 was not increased and IL-15 was reduced ($p < 0.02$). The IFN-gamma inducing cytokine, IL-18 was increased in tumour-bearing liver ($p < 0.02$), while pro-inflammatory TNF-alpha was suppressed ($p < 0.05$). These results suggest that, whilst there is a significant inflammatory immune response in tumour-bearing liver, evidenced by increased levels of IFN-gamma, disproportionate increase in IL-10 may be a key factor in facilitating tumour progression. Therapies aimed at antagonising IL-10-mediated immunosuppression may prove a useful strategy in the future treatment of metastatic disease. (c) 2006 Elsevier Ltd. All rights reserved.

L6 ANSWER 9 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2006:335729 BIOSIS <<LOGINID::20100316>>
DN PREV200600335695
TI Disseminated herpes zoster in diabetes mellitus.
Original Title: Herpes zoster generalisatus bei diabetes mellitus.
AU Graue, N.; Grabbe, S.; Dissemond, J. [Reprint Author]
CS Univ Klinikum Essen, Klin and Poliklin Dermatol Venerol and Allergol, Hufelandstr 55, D-45147 Essen, Germany
joachimdissemond@hotmail.com
SO DMW Deutsche Medizinische Wochenschrift, (FEB 24 2006) Vol. 131, No. 8,

pp. 384-386.
CODEN: DDMWDF. ISSN: 0012-0472.
DT Article
LA German
ED Entered STN: 5 Jul 2006
Last Updated on STN: 5 Jul 2006
AB History and admission findings: A 71-year old man presented with painful hemorrhagic vesicles and papules over the entire body that had persisted for three days. Type 2 diabetes mellitus type 2 had been diagnosed 20 years ago and had not been treated for the last 5 years. Therapy had been discontinued by the patient.Investigations: HbA1c (11,9%) and blood glucose levels (up to 360 mg/dl) were abnormal. Varicella-zoster-DNA was replicated by PCR from the vesicle fluid.Diagnosis and treatment: After the clinical diagnosis of disseminated herpes zoster had been confirmed systemic therapy with aciclovir 10 mg/kg day was started. There was no evidence of malignancy. Insulin therapy was intiated.Conclusion: Dissemination is a rare complication of herpes zoster, aided by immunosupression. In the presented case there was no evidence of malignancy or other cause of immunosuppression, but the patient also had type 2 diabetes with very high blood glucose levels. The diabetes was thought to be causally related to the ***ineffective*** ***immune*** ***response*** to varicella zoster virus. There has been no previus published report of this relationship.

L6 ANSWER 10 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2006:79965 BIOSIS <<LOGINID::20100316>>
DN PREV200600086706
TI Premalignant disease characterization in a mouse model of infectious liver cancer.
AU Rogers, Arlin B.; Whary, Mark T.; Sundina, Nataliya; Boutin, Samuel R.; Fox, James G.
SO Gastroenterology, (APR 2004) Vol. 126, No. 4, Suppl. 2, pp. A740.
Meeting Info.: Digestive Disease Week/105th Annual Meeting of the American-Gastroenterological-Association. New Orleans, LA, USA. May 16 -20, 2004. Amer Gastroenterol Assoc.
CODEN: GASTAB. ISSN: 0016-5085.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 25 Jan 2006
Last Updated on STN: 25 Jan 2006
AB We used a natural murine model of infectious liver cancer, Helicobacter hepaticus in A/JCr mice, to investigate in vivo pathogenesis of inflammation-associated hepatocarcinogenesis in the premalignant period. We inoculated pregnant dams at conception or mid-gestation, and pups at 3 or 12 weeks of age, with H. hepaticus or vehicle only, and collected offspring at 3, 6, or 12 months of age. Assays performed included PCR, hemogram, serum chemistry, systemic and mucosal antibody ELISA, restimulated splenocyte cytokine ELISA, histopathology with lesion grading, special stains, and immumohistochemistry. Mice born to dams infected intragestationally, with repeat inoculation of offspring postnatally, exhibited a trend towards more severe and accelerated disease, while those inoculated only at 12 weeks of age were resistant. Male mice exhibited much greater risk than females for severe hepatitis and preneoplasia; however, only about half of males were affected. Affected male mice displayed chronic active hepatitis and preneoplastic

foci of altered hepatocytes, while females were more likely to develop only persistent hepatitis. All infected mice maintained high fecal bacterial loads, but intrahepatic bacteria were restricted to those with active hepatitis. Serum AST and ALT levels were increased in infected mice, but not correlated with hepatitis severity. Peak serum IgG2a (Th1) preceded IgG1 (Th2) antibody titers, and females mounted the strongest mucosal IgA responses, but no antibody or restimulated splenocyte cytokine measure correlated with hepatitis grade. Immunohistochemistry identified leukocyte subsets in lobular lesions and portal tertiary lymphoid nodules, and demonstrated intraphagocytic iNOS and hepatocellular COX-2 but not caspase-3 upregulation. Special stains demonstrated diastase-resistant PAS + material but not iron within phagocytes, and intracanalicular bacteria adjacent to active lobular lesions. In conclusion, early and repeated inoculation of A/JCr mice with H. hepaticus accentuates hepatitis and preneoplastic progression. Males are prone to the most severe disease, but display a bipolar pattern of susceptibility, Intrahepatic inflammation is driven by local signals, and is characterized by a vigorous but ultimately ***ineffective*** ***immune*** ***response*** . Continued study of H. hepaticus infection in A/JCr mice will help to clarify host factors associated with disease susceptibility, and may point to new targets for the intervention of hepatitis-associated liver cancer in humans.

L6 ANSWER 11 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2006:77567 BIOSIS <<LOGINID::20100316>>
DN PREV200600084308
TI Interleukin-10 suppresses Helicobacter pylori induced interleukin-8 secretion and increases H. pylori survival by acting directly on the epithelial cell.
AU Robinson, Karen; Atherton, John
SO Gastroenterology, (APR 2004) Vol. 126, No. 4, Suppl. 2, pp. A261. Meeting Info.: Digestive Disease Week/105th Annual Meeting of the American-Gastroenterological-Association. New Orleans, LA, USA. May 16 -20, 2004. Amer Gastroenterol Assoc.
CODEN: GASTAB. ISSN: 0016-5085.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 25 Jan 2006
Last Updated on STN: 25 Jan 2006
AB Purpose: Elevated levels of the anti-inflammatory cytokine IL-10 are induced in the human gastric mucosa by H. pylori infection. The infection provokes a vigorous but ***ineffective*** ***immune*** ***response*** and colonisation may persist life-long. Some individuals develop peptic ulceration or adenocarcinoma, but infection usually causes asymptomatic chronic gastritis, implying that inflammation is down-regulated to avoid serious pathology. Experiments were therefore conducted to determine whether IL-10 could function to suppress the inflammatory response of human gastric epithelial cells, induced by incubation with H. pylori in vitroMethods: Recombinant human IL-10 (at 20 ng/ml) was added to AGS cell monolayers in the presence of 2 X 10(8) cfu/ml H. pylori strain 60190 (cag Pal+). Following incubation for up to 24 hours, supernatants were assayed for IL-8 by ELISA, and flow cytometry was used to examine AGS cells for ICAM1 expression. Western blotting was

used to quantify COX-2 protein expression in AGS cell lysates,Results: The addition of rhIL-10 to co-cultures of AGS cells and H. pylon significantly inhibited IL-8 secretion by up to 50% and ICAM1 expression by 15%, but COX-2 expression levels were unaffected. Significantly higher concentrations of bacteria were recovered from the co-cultures to which rhIL-10 was added, but IL-10 did not improve the growth of H. pylon alone.Conclusions: IL-10 downregulates H. pylon-induced epithelial cell IL-8 expression, possibly contributing to suppression of H. pylori-associated inflammation in vivo. More interestingly, it increases H. pylon survival during epithelial co-culture. This suggests that the increases in bacterial density observed in animal models with enhanced Th2 responses may be due to effects at the level of the epithelial cell. We are now investigating whether this is through reduced expression of anti-bacterial peptides.

L6 ANSWER 12 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2005:477424 BIOSIS <<LOGINID::20100316>>
DN PREV200510269328
TI Highly efficient presentation of endogenously processed class I peptides by artificial APC for the generation of effective anti-tumor responses.
AU Hirano, Naoto [Reprint Author]; Butler, Marcus O.; Xia, Zhinan; Nadler, Lee M.
CS Dana Farber Canc Inst, Dept Med Oncol, Boston, MA 02115 USA
SO Blood, (NOV 16 2004) Vol. 104, No. 11, Part 1, pp. 381A.
Meeting Info.: 46th Annual Meeting of the American-Society-of-Hematology. San Diego, CA, USA. December 04 -07, 2004. Amer Soc Hematol.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Conference; (Meeting)
Conference; (Meeting Poster)
LA English
ED Entered STN: 16 Nov 2005
Last Updated on STN: 16 Nov 2005
AB Appropriate processing and presentation of tumor associated antigens (TAA) by antigen presenting cells (APC) is absolutely required for the development of clinically relevant anti-tumor T cell responses. One common approach which utilizes the exogenous pulsing of synthetic peptides onto APC can sometimes generate ***ineffective*** ***immune*** ***responses*** . This failure may, in part, be the consequenceof erroneous conformations of HLA/synthetic pulsed peptides which can differ from conformations formed with FILA/endogenous peptides that are derived from intracellular proteins. Also, endogenously processed peptides sometimes undergo post-translational modifications during transport to the cell surface, a process that does not occur with exogenously loaded peptides. Since our goal is to induce immunity that can recognize TAA that are endogenously presented by tumors, it is logical that the ideal APC would not only express the required immunoaccessory molecules, but would also endogenously process and appropriately present target antigenic peptides. In this report, we employed our artificial APC (aAPC) that expresses HLA-A2, CD80, and CD83 and is capable of priming andsupporting the prolonged expansion of peptide specific CD8+ cytotoxic T cells(CTL). We hypothesized that aAPC can endogenously process and present HLA class I peptides and can induce functional T cell immunity. To test this, aAPC was transduced with an EGFP-mini MP1 (aa 55-66) fusion gene containing the sequence for the influenza derived peptide MP58 (aa 58-66). We observed that this HLA-A2 restricted peptide is processed and presented by aAPC by demonstrating that MP58 specific CTL are able to recognize aAPC/mini MP I

target cells. This was completely abrogated by treating aAPC/mini MP1 with proteasome inhibitors,suggesting that MP58 is endogenously processed by the proteasome. This was confirmed by the elevation of EGFP-mini MP1 fusion protein and the accumulation of ubiquitinated forms as detected by flow cytometry and Western blot analysis, respectively. At the protein level, aAPC was shown to express all proteasomesubunits examined and to upregulate immunoproeosome subunits with exposure toIFN-gamma. We biochemically confirmed the presence of MP58 in the A2 groove on the surface of aAPC/mini MP1, by performing reverse phased HPLC, mass spectrometry and peptide sequencing of peptides directly acid stripped from the cellsurface. Since aAPC expresses only one HLA allele, A2, this finding provides strong support that MP58 is processed and presented in the groove of the A2 molecule on aAPC/mini MP1. We next evaluated the density of MP58 presented by HLA-A2 on aAPC/mini MP1. A2 positive CD8+ T cells were stimulated at weekly intervals by either aAPC/mini MP1 or parental aAPC exogenously pulsed with graded concentrations of synthetic MP58. After three stimulations, peptide specificity of generated CTL was examined by tetramer analysis. The comparison of tetramer staining revealed that the density of endogenously processed and presented MP58 corresponded to pulsing aAPC with 100 mu g/ml. In order to extend this strategy to a TAA, we transduced aAPC with EGFP-mini MART1 (aa 27-35) mini gene.We have demonstrated that MART1 peptide is processed and presented on the cell surface and have induced the expansion of MART1 peptide specific T cells. These results suggest that our APC can naturally process and present class I restricted peptides, resulting in the efficient priming and expansion of clinically relevant antigen specific CTL.

L6 ANSWER 13 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2005:348618 BIOSIS <<LOGINID::20100316>>
DN PREV200510138622
TI Helicobacter pylori-induced macrophage apoptosis requires activation of ornithine decarboxylase by c-Myc.
AU Cheng, Yulan; Chaturvedi, Rupesh; Asim, Mohammad; Bussiere, Francoise I.; Xu, Hangxiu; Casero, Robert A. Jr.; Wilson, Keith T. [Reprint Author]
CS Univ Maryland, Sch Med, Greenbaum Canc Ctr, 22 S Greene St,Rm N3W62, Baltimore, MD 21201 USA
kwilson@umaryland.edu
SO Journal of Biological Chemistry, (JUN 10 2005) Vol. 280, No. 23, pp. 22492-22496.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 8 Sep 2005
Last Updated on STN: 8 Sep 2005
AB Helicobacter pylori infection causes chronic inflammation of the gastric mucosa that results from an ***ineffective*** ***immune*** ***response*** . We have demonstrated that one underlying mechanism is induction of macrophage apoptosis mediated by polyamines. The transcription factor c-Myc has been linked to induction of ornithine decarboxylase (ODC), the rate-limiting enzyme in polyamine synthesis. We determined whether H. pylori stimulates transcriptional activation of ODC in macrophages, whether this occurs via c-Myc, and whether these events regulate activation of apoptosis. H. pylori induced a significant increase in ODC promoter activity that peaked at 6 h after stimulation and was closely paralleled by similar increases in ODC mRNA, protein, and enzyme activity. By 2 h after stimulation, c-Myc mRNA and protein

expression was induced, protein was translocated to the nucleus, and there was specific binding of a consensus probe for c-Myc to nuclear extracts. Both an antennapedia- linked inhibitor of c-Myc binding (Int-H1-S6A, F8A) and transfection of a c-Myc dominant-negative construct significantly attenuated H. pyloriinduced ODC promoter activity, mRNA, enzyme activity, and apoptosis in parallel. Transfection of ODC small interfering RNA inhibited ODC activity and apoptosis to the same degree as inhibition of c-Myc binding. These studies indicate that c-Myc is an important mediator of macrophage activation and may contribute to the mucosal inflammatory response to pathogens such as H. pylori by its effect on ODC.

L6 ANSWER 14 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2005:152884 BIOSIS <<LOGINID::20100316>>
DN PREV200500151956
TI Host adaptation and immune modulation are mediated by homologous recombination in Helicobacter pylori.
AU Robinson, Karen [Reprint Author]; Loughlin, Michael F.; Potter, Rebecca; Jenks, Peter J.
CS Queens Med CtrInst Infect Immun and Inflammat, Univ Nottingham, C Floor,W Block, Nottingham, NG7 2UH, UK
karen.robinson@nottingham.ac.uk
SO Journal of Infectious Diseases, (February 15 2005) Vol. 191, No. 4, pp. 579-587. print.
CODEN: JIDIAQ. ISSN: 0022-1899.
DT Article
LA English
ED Entered STN: 20 Apr 2005
Last Updated on STN: 20 Apr 2005
AB Rearrangement of genomic DNA via homologous recombination provides an alternative mechanism of gene regulation that is essential for successful colonization of the gastric mucosa by Helicobacter pylori. Inoculation of outbred mice with the H. pylori SS1 wild-type strain elicited a T helper (Th) 2 response and established a persistent infection. In contrast, inoculation with an isogenic H. pylori strain defective for homologous recombination elicited a Th1-mediated immune response and clearance of infection within 70 days. We, therefore, demonstrate that recombination is critical for mediating persistence of a microbial pathogen through the induction of ***ineffective*** ***immune*** ***responses*** .

L6 ANSWER 15 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2005:90066 BIOSIS <<LOGINID::20100316>>
DN PREV200500087921
TI Activation of dendritic cells that cross-present tumor-derived antigen licenses CD8+ CTL to cause tumor eradication.
AU van Mierlo, Geertje J. D.; Boonman, Zita F. H. M.; Dumortier, Helene M. H.; den Boer, Annemieke Th.; Fransen, Marieke F.; Nouta, Jan; van der Voort, Ellen I. H.; Offringa, Rienk; Toes, Rene E. M.; Melief, Cornelis J. M. [Reprint Author]
CS Med CtrDept Immunhematol and Bloodtransuf, Leiden Univ, Postal Zone E3-Q,Albinusdreef 2, NL-2333 ZA, Leiden, Netherlands
C.Melief@lumc.nl
SO Journal of Immunology, (December 1 2004) Vol. 173, No. 11, pp. 6753-6759. print.
ISSN: 0022-1767 (ISSN print).
DT Article

LA English
ED Entered STN: 2 Mar 2005
Last Updated on STN: 2 Mar 2005
AB The fate of naive CD8+ T cells is determined by the environment in which they encounter MHC class I presented peptide Ags. The manner in which tumor Ags are presented is a longstanding matter of debate. Ag presentation might be mediated by tumor cells in tumor, draining lymph nodes or via cross-presentation by professional APC. Either pathway is insufficient to elicit protective antitumor immunity. We now demonstrate using a syngeneic mouse tumor model, expressing an Ag derived from the early region 1A of human adenovirus type 5, that the inadequate nature of the antitumor CTL response is not due to direct Ag presentation by the tumor cells, but results from presentation of tumor-derived Ag by nonactivated CD11c+ APC. Although this event results in division of naive CTL in tumor draining lymph nodes, it does not establish a productive immune response. Treatment of tumor-bearing mice with dendritic cell-stimulating agonistic anti-CD40 mAb resulted in systemic efflux of CTL with robust effector function capable to eradicate established tumors. For efficacy of anti-CD40 treatment, CD40 ligation of host APC is required because adoptive transfer of CD40-proficient tumor-specific TCR transgenic CTL into CD40-deficient tumor-bearing mice did not lead to productive antitumor immunity after CD40 triggering in vivo. CpG and detoxified LPS (MPL) acted similarly as agonistic anti-CD40 mAb with respect to CD8+ CTL efflux and tumor eradication. Together these results indicate that dendritic cells, depending on their activation state, orchestrate the outcome of CTL-mediated immunity against tumors, leading either to an ***ineffective*** ***immune*** ***response*** or potent antitumor immunity.

L6 ANSWER 16 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2004:438650 BIOSIS <<LOGINID::20100316>>
DN PREV200400437474
TI Cytokine and inducible nitric oxide synthase mRNA expression during experimental murine cryptococcal meningoencephalitis.
AU Maffei, Claudia M. L.; Mirels, Laurence F.; Sobel, Raymond A.; Clemons, Karl V. [Reprint Author]; Stevens, David A.
CS Dept MedDiv Infect Dis, Santa Clara Valley Med Ctr, 751 S Bascom Ave, San Jose, CA, 95128, USA
clemons@cimr.org
SO Infection and Immunity, (April 2004) Vol. 72, No. 4, pp. 2338-2349. print.
ISSN: 0019-9567 (ISSN print).
DT Article
LA English
ED Entered STN: 17 Nov 2004
Last Updated on STN: 17 Nov 2004
AB The immune events that take place in the central nervous system (CNS) during cryptococcal infection are incompletely understood. We used competitive reverse transcription-PCR to delineate the time course of the local expression of mRNAs encoding a variety of cytokines and inducible nitric oxide synthase (iNOS) during progressive murine cryptococcal meningoencephalitis and assessed the CNS inflammatory response using immunohistochemistry. Interleukin 18 (IL-18), transforming growth factor beta1, and IL-12p40 mRNAs were constitutively expressed in the brains of infected and uninfected mice; IL-2 mRNA was not detected at any time. Increased levels of transcripts corresponding to IL-1alpha, tumor necrosis factor alpha (TNF-alpha), and NOS were detected as early as day 1

postinfection, with TNF-alpha rising by apprx30-fold and MOS increasing by apprx5-fold by day 7. Each remained at these levels thereafter. IL-4, IL-6, and gamma interferon transcripts were detected on day 5, and IL-1beta and IL-10 transcripts were detected beginning on day 7. Once detected, each remained at a relatively constant level through 28 days of infection. This cytokine profile does not suggest a polarized Th1 or Th2 response. Immunohistochemistry did not reveal inflammatory infiltrates before day 7, despite the presence of cryptococci. Intraparenchymal abscesses with inflammatory cells in their peripheries were found beginning on day 10. The infiltrates were comprised primarily of cells expressing CD4, CD8, or CD11b; low numbers of cells expressing CD45R/B220 were also present. The persistence of Cryptococcus observed in the CNS may result from an ***ineffective*** ***immune*** ***response*** , perhaps owing to an insufficient anticryptococcal effector function of endogenous glial cells resulting from competing pro- and anti-inflammatory cytokines. These data detail the immune response in the brain and could be important for the future design of specific immunomodulatory therapies for this important opportunistic infection.

L6 ANSWER 17 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2003:340045 BIOSIS <<LOGINID::20100316>>
DN PREV200300340045
TI Immunoglobulin G subisotype responses of pneumonic and healthy, exposed foals and adult horses to Rhodococcus equi virulence-associated proteins.
AU Hooper-McGrevy, Kathleen E.; Wilkie, Bruce N.; Prescott, John F. [Reprint Author]
CS Department of Pathobiology, University of Guelph, Guelph, ON, N1G 2W1, Canada
prescott@uoguelph.ca
SO Clinical and Diagnostic Laboratory Immunology, (May 2003) Vol. 10, No. 3, pp. 345-351. print.
ISSN: 1071-412X (ISSN print).
DT Article
LA English
ED Entered STN: 23 Jul 2003
Last Updated on STN: 23 Jul 2003
AB Rhodococcus equi causes severe pyogranulomatous pneumonia in foals and in immunocompromised humans. Replication of virulent isolates within macrophages correlates with the presence of a large plasmid which encodes a family of seven virulence-associated proteins (VapA and VapC to VapH), whose functions are unknown. Although cell-mediated immunity is thought to be crucial in eliminating R. equi infection, antibody partially protects foals. The antibody response to both VapA and VapC was similar in six adult horses and six naturally exposed but healthy foals, as well as in eight foals with R. equi pneumonia. The immunoglobulin G (IgG) subisotype response of pneumonic foals to Vap proteins was significantly IgGb biased and also had a trend toward higher IgGT association compared to the isotype association of antibody in adult horses and healthy exposed foals. This suggests that in horses, IgGb and IgGT are Th2 isotypes and IgGa is a Th1 isotype. Furthermore, it suggests that foals which develop R. equi pneumonia have a Th2-biased, ***ineffective*** ***immune*** ***response*** whereas foals which become immune develop a Th1-biased immune response. Pneumonic foals had significantly more antibody to VapD and VapE than did healthy exposed foals. This may indicate a difference in the expression of these two Vap proteins during persistent infection. Alternatively, in pneumonic foals the deviation of the immune response

toward VapD and VapE may reflect a bias unfavorable to R. equi resistance. These data indicate possible age-related differences in the equine immune response affecting Th1-Th2 bias as well as antibody specificity bias, which together favor the susceptibility of foals to R. equi pneumonia.

L6 ANSWER 18 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2002:516227 BIOSIS <<LOGINID::20100316>>
DN PREV200200516227
TI Hepatitis C virus core protein leads to immune suppression and liver damage in a transgenic murine model.
AU Soguero, Carolina; Joo, Myungsoo; Chianese-Bullock, Kimberly A.; Nguyen, Duong Tony; Tung, Kenneth; Hahn, Young S. [Reprint author]
CS Department of Microbiology, University of Virginia, HSC Box 801386, Charlottesville, VA, 22908, USA
ysh5e@virginia.edu
SO Journal of Virology, (September, 2002) Vol. 76, No. 18, pp. 9345-9354. print.
CODEN: JOVIAM. ISSN: 0022-538X.
DT Article
LA English
ED Entered STN: 2 Oct 2002
Last Updated on STN: 2 Oct 2002
AB Hepatitis C virus (HCV) is remarkably efficient in establishing persistent infection, possibly mediated by an impaired immune response to HCV infection. There is compelling evidence that HCV can infect immune cells, such as macrophages, B cells, and T cells. It has been previously reported that HCV core, the first protein expressed during the early phase of viral infection, contains the immunomodulatory function of suppressing host immune responses. This altered function of immune cells caused by HCV infection may explain the ***ineffective*** ***immune*** ***response*** to HCV. To further characterize the immunomodulatory role of HCV core in vivo, we generated transgenic (TG) mice by directing the expression of core protein to T lymphocytes by using the CD2 promoter. T-lymphocyte responses, including the production of gamma interferon and interleukin-2, were significantly diminished in these mice compared to their non-TG littermates. The inhibition of T-lymphocyte responsiveness may be due to the increased susceptibility of peripheral T lymphocytes to Fas-mediated apoptosis. Surprisingly, significant lymphocyte infiltration was observed in the portal tracts of livers isolated from core TG mice, associated with increasing serum alanine aminotransferase levels. Moreover, no intrahepatic lymphocytes or liver damage was found in non-TG littermates and core TG mice bred to Fas-deficient lpr mice. These results suggest that HCV core drives liver injury by increasing Fas-mediated apoptosis and liver infiltration of peripheral T cells.

L6 ANSWER 19 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2002:461568 BIOSIS <<LOGINID::20100316>>
DN PREV200200461568
TI A Tat subunit vaccine confers protective immunity against the immune-modulating activity of the human immunodeficiency virus type-1 Tat protein in mice.
AU Agwale, S. M.; Shata, M. T.; Reitz, M. S., Jr.; Kalyanaraman, V. S.; Gallo, R. C.; Popovic, M.; Hone, D. M. [Reprint author]
CS Division of Vaccine Research, University of Maryland Biotechnology Institute, Baltimore, MD, 21202, USA

hone@umbi.umd.edu
SO Proceedings of the National Academy of Sciences of the United States of America, (July 23, 2002) Vol. 99, No. 15, pp. 10037-10041. print. CODEN: PNASA6. ISSN: 0027-8424.
DT Article
LA English
ED Entered STN: 28 Aug 2002
Last Updated on STN: 28 Aug 2002
AB The rational design of new therapies against HIV-1 necessitates an improved understanding of the mechanisms underlying the production of ***ineffective*** ***immune*** ***responses*** to HIV-1 in most infected individuals. This report shows that the CD8+ T cell responses to gp120 were greatly diminished in mice vaccinated with a bicistronic gp120-Tat DNA vaccine, compared with those induced by a DNA vaccine encoding gp120 alone. The CD8+ T cell responses induced by the latter included strong gp120-specific IFN-gamma secretion and protective antiviral immunity against challenge by a vaccinia-env pseudotype. The degree to which Tat influenced CD8+ T cell responses depended on the bioactivity of Tat. Thus, a bicistronic DNA vaccine that expresses gp120 and a truncated Tat defective for LTR activation elicited strong IFN-gamma-secreting CD8+ T cell responses to gp120 but conferred only marginal protection against the vaccinia-env challenge. The effect of Tat was completely blocked, however, by immunization with inactivated Tat protein before vaccination with the bicistronic gp120-Tat DNA vaccine.

L6 ANSWER 20 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2002:418891 BIOSIS <<LOGINID::20100316>>
DN PREV200200418891
TI CD3-zetachain expression of intratumoral lymphocytes is closely related to survival in gastric carcinoma patients.
AU Ishigami, Sumiya [Reprint author]; Natsugoe, Shoji; Tokuda, Koki; Nakajo, Akihiro; Higashi, Hiroshi; Iwashige, Hirohumi; Aridome, Kuniaki; Hokita, Shuichi; Aikou, Takashi
CS First Department of Surgery, Kagoshima University School of Medicine, 8-35-1 Sakuragaoka, Kagoshima, 890, Japan
ishiga@med2.kufm.kagoshima-u.ac.jp
SO Cancer, (March 1, 2002) Vol. 94, No. 5, pp. 1437-1442. print. CODEN: CANCAR. ISSN: 0008-543X.
DT Article
LA English
ED Entered STN: 7 Aug 2002
Last Updated on STN: 7 Aug 2002
AB BACKGROUND: Impaired or reduced CD3 zeta chain (CD3-zeta) expression in T cells has been identified in various cancers and may be associated with an ***ineffective*** ***immune*** ***response*** . The clinical significance of CD3-zeta chain expression in tumor-infiltrating lymphocytes (TILs) in gastric carcinoma remains unclear. METHODS: The authors immunohistochemically investigated CD3-zeta expression in TILs in 185 patients who had undergone curative gastrectomy. CD3-zeta/CD3 epsilon (CD3-epsilon) ratios were calculated. Patients were divided into two groups: a normal CD3-zeta group (n=121) and a reduced CD3-zeta group (n=64). Patients with a zeta per epsilon ratio of greater than 66% were placed in the normal CD3-zeta group. RESULTS: Patients in the normal CD3-zeta group had fewer lymph node metastasis (P<0.01) and a shallower depth of invasion (P<0.05) than those in the reduced CD3-zeta group. The 5-year survival rate was 72% in the normal CD3-zeta group, which was

significantly better than that in the reduced CD3-zeta group (55%; P<0.01). When stratified according to clinical stage, the prognostic value was significantly different only in Stage IV patients. Multivariate analysis showed that CD3-zeta expression was an independent prognostic factor (P=0.03) next to depth of invasion and lymph node involvement. CONCLUSIONS: Reduced CD3-zeta expression in TILs was strongly correlated with progressive disease in gastric carcinomas. CD3-zeta expression in TILs is considered an immunologic, independent prognostic marker in gastric carcinoma patients. CD3-zeta normalization with cytokine treatment may lead to prolonged survival in advanced gastric carcinoma patients.

L6 ANSWER 21 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2002:99223 BIOSIS <<LOGINID::20100316>>
DN PREV200200099223
TI Immunoglobulin genes expressed by B-lymphocytes infiltrating cervical carcinomas show evidence of antigen-driven selection.
AU O'Brien, Philippa M. [Reprint author]; Tsirimonaki, Emmanouella; Coomber, David W. J.; Millan, David W. M.; Davis, Jonathon A.; Campo, M. Saveria
CS Department of Veterinary Pathology, University of Glasgow, Bearsden Road, Glasgow, G61 1QH, UK
p.obrien@vet.gla.ac.uk
SO Cancer Immunology Immunotherapy, (December, 2001) Vol. 50, No. 10, pp. 523-532. print.
CODEN: CIIMDN. ISSN: 0340-7004.
DT Article
LA English
ED Entered STN: 24 Jan 2002
Last Updated on STN: 25 Feb 2002
AB Lymphocyte infiltration is often present in cervical cancer lesions, possibly reflecting an ongoing (but ***ineffective***) ***immune*** ***response*** to the tumour. B-lymphocytes are the predominant lymphocyte infiltrate in pre-malignant cervical lesions, where they are thought to comprise the host immune response to active human papillomavirus (HPV) infection. Although B cells are less frequently detected in cervical tumours, a high proportion of terminally differentiated plasma cells expressing tumour-specific immunoglobulin (Ig) remain. The antigen specificity and functional significance of the antibody response to cervical tumours is unknown. As part of a study to characterise the antibodies expressed by the tumour-infiltrating B cells (TIL-B) in cervical tumours using antibody phage display, we examined expressed Ig gene sequences to determine if there was molecular evidence of a selective response to antigenic changes in the transformed epithelial cells. We found that biased variable region gene usage by the B cells and the rate of somatic hypermutation in the rearranged Ig heavy chain variable regions (VH) both indicated antigenic selection of the B cells. We also found evidence of affinity maturation, as indicated by the detection of antibodies of the IgG1, IgG2 and IgA isotypes, and possible clonal selection of the Ig receptors. These data support the notion that B-lymphocytes and plasma cells infiltrating cervical carcinomas are the result of an antigen-induced response to HPV infection or transformation.

L6 ANSWER 22 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2001:195037 BIOSIS <<LOGINID::20100316>>
DN PREV200100195037

TI Mucosal immunity in the lung and upper airway.
AU Kyd, Jennelle M. [Reprint author]; Foxwell, A. Ruth; Cripps, Allan W.
CS Division of Science and Design, Gadi Research Centre, University of Canberra, Canberra, ACT, 2601, Australia
kyd@scides.canberra.edu.au
SO Vaccine, (21 March, 2001) Vol. 19, No. 17-19, pp. 2527-2533. print.
CODEN: VACCDE. ISSN: 0264-410X.
DT Article
LA English
ED Entered STN: 20 Apr 2001
Last Updated on STN: 18 Feb 2002
AB The mucosal surfaces of the lungs and upper airways are common sites for infection. Extensive studies of the mechanisms associated with immune responses in the respiratory tract have found that understanding the system is challenging and involves many complex interactions to prevent and eliminate infection. Immune protection against diseases transmitted through the respiratory tract requires an understanding of the important aspects associated with beneficial, detrimental or ***ineffective*** ***immune*** ***responses*** . Two critical aspects of an immune response against a pathogen are that of the inductive stage, either induced by vaccination or primary infection, and the effector stage, the ability to recognise, respond to and eliminate the infection without detriment to the host. An immunisation strategy must not only have a measure of the induced antigen specific response, but this response must also be protective.

L6 ANSWER 23 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2000:349508 BIOSIS <<LOGINID::20100316>>
DN PREV200000349508
TI Ineffective cellular immune response associated with T-cell apoptosis in susceptible Mycobacterium bovis BCG-infected mice.
AU Kremer, Laurent; Estaquier, Jerome [Reprint author]; Wolowczuk, Isabelle; Biet, Franck; Ameisen, Jean-Claude; Locht, Camille
CS INSERM E9922, Groupe Hospitalier Bichat-Claude Bernard, 16 Rue Henri Huchard, 75018, Paris, France
SO Infection and Immunity, (July, 2000) Vol. 68, No. 7, pp. 4264-4273. print.
CODEN: INFIBR. ISSN: 0019-9567.
DT Article
LA English
ED Entered STN: 16 Aug 2000
Last Updated on STN: 7 Jan 2002
AB It has previously been reported that inhibition of delayed-type hypersensitivity-mediating functions of T cells during mycobacteria infection in mice is haplotype dependent. In the present study, we show that Mycobacterium bovis BCG infection induced, in susceptible C57BL/6 and BALB/c mice but not in resistant C3H/HeJ and DBA/2 mice, an important splenomegaly. An in vitro defect in T-cell proliferation in response to T-cell receptor (TCR) stimulation with mitogens or anti-CD3 antibodies was associated with enhanced levels of CD4+ and CD8+ T-cell apoptosis in susceptible but not in resistant mice 2 weeks after infection. Further investigations of C57BL/6 and C3H/HeJ mice revealed that in vivo splenomegaly was associated with destruction of the lymphoid tissue architecture, liver cellular infiltrates, and increased numbers of apoptotic cells in both spleen and liver tissue sections. Infection of C57BL/6 mice but not of C3H/HeJ mice induced massive production of tumor necrosis factor alpha (TNF-alpha) in serum, as well as an increase in Fas

and Fas ligand (FasL) expression in T cells. In vitro addition of neutralizing anti-TNF-alpha antibodies led to a significant reduction in CD3-induced T-cell apoptosis of both CD4+ and CD8+ T cells of C57BL/6 mice, while the blockade of Fas-FasL interactions reduced apoptosis only in CD4+ but not in CD8+ T cells. Together, these results suggest that TNF-alpha and Fas-FasL interactions play a role in the activation-induced cell death (AICD) process associated with a defect in T-cell proliferation of the susceptible C57BL/6 mice. T-cell death by apoptosis may represent one of the important components of the ***ineffective*** ***immune*** ***response*** against mycobacterium-induced immunopathology in susceptible hosts.

L6 ANSWER 24 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2000:149248 BIOSIS <<LOGINID::20100316>>
DN PREV200000149248
TI Immune response to Trichinella spiralis larvae after treatment with the anti-allergic compound ketotifen.
AU Doligalska, Maria [Reprint author]
CS Department of Parasitology, Institute of Zoology, University of Warsaw, Krakowskie Przedmiescie 26/28, PL-00927, Warsaw, Poland
SO Parasitology Research, (March, 2000) Vol. 86, No. 3, pp. 232-238. print.
CODEN: PARREZ. ISSN: 0932-0113.
DT Article
LA English
ED Entered STN: 19 Apr 2000
Last Updated on STN: 4 Jan 2002
AB Ketotifen was used as an anti-allergic agent to study the relationship between eosinophil-related responses and IgG1 and IgG2a antibody responses in BALB/c mice infected with Trichinella spiralis. The results showed that leukocyte and eosinophil numbers and interleukin-5 (IL-5) concentrations in the peritoneal fluid increased after exposure to nematodes and the increases were slightly greater in animals treated with ketotifen. A decreased concentration of eosinophil peroxidase and an elevation in IgG1 accompanied the muscle phase of infection. In mice treated with ketotifen, antibody-mediated recognition of muscle larvae was delayed. The retardation of IgG1 and IgG2a responses may have been responsible for the ***ineffective*** ***immune*** ***response*** against larvae migrating into the muscle. The activation of eosinophils was accompanied by changes in IL-5 concentration, but these changes were not associated with differences in protection against infection.

L6 ANSWER 25 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2000:114183 BIOSIS <<LOGINID::20100316>>
DN PREV200000114183
TI Therapeutic immunization against Helicobacter pylori infection in the absence of antibodies.
AU Sutton, Philip [Reprint author]; Wilson, John; Kosaka, Tadashi; Wolowczuk, Isabelle; Lee, Adrian
CS School of Microbiology and Immunology, University of New South Wales, Sydney, NSW, 2052, Australia
SO Immunology and Cell Biology, (Feb., 2000) Vol. 78, No. 1, pp. 28-30. print.
CODEN: ICBIEZ. ISSN: 0818-9641.

DT Article
LA English
ED Entered STN: 29 Mar 2000
Last Updated on STN: 3 Jan 2002
AB Helicobacter pylori is an important human pathogen. Prophylactic immunization with bacterial antigen plus an adjuvant protects mice against challenge with live H. pylori. Surprisingly, it was found that immunizations of mice already infected with Helicobacter also influenced bacterial colonization. This concept of therapeutic immunization is a novel phenomenon. Because H. pylori lives in the lumen of the stomach, it was initially hypothesized that the protective mechanism would involve induction of secretory IgA. However, work with knockout mice has demonstrated that prophylactic immunization is equally effective in mice deficient in IgA and even in muMT mice lacking B lymphocytes. Currently nothing is known about therapeutic vaccination and the effect of immunizing a host with an ongoing ***ineffective*** ***immune*** ***response*** . To address this, we infected B-cell deficient, muMT mice with H. pylori and therapeutically immunized them four times in 3 weeks with bacterial sonicate and cholera toxin adjuvant. These immunizations significantly reduced colonization by H. pylori. The antibody-negative status of the muMT mice was confirmed by ELISA. Thus, therapeutic immunization stimulates an immune response, which reduces H. pylori infection via a mechanism that is antibody independent. How this is achieved remains to be determined, but may well involve a novel immune mechanism.

L6 ANSWER 26 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 1999:171784 BIOSIS <<LOGINID::20100316>>
DN PREV199900171784
TI Expression of CD3-zeta on T-cells in primary cervical carcinoma and in metastasis-positive and -negative pelvic lymph nodes.
AU De Gruijl, T. D.; Bontks, H. J.; Peccatori, F.; Gallee, M. P. W.; Helmerhorst, T. J. M.; Verheijen, R. H. M.; Aarbiou, J.; Mulder, W. M. C.; Walboomers, J. M. M.; Meijer, C. J. L. M.; Van De Vange, N.; Scheper, R. J. [Reprint author]
CS Dep. Pathol., Free Univ. Hosp., P.O. Box 7057, 1007 MB Amsterdam, Netherlands
SO British Journal of Cancer, (March, 1999) Vol. 79, No. 7-8, pp. 1127-1132. print.
CODEN: BJCAAI. ISSN: 0007-0920.
DT Article
LA English
ED Entered STN: 19 Apr 1999
Last Updated on STN: 19 Apr 1999
AB Lymphocytic infiltrate is often present in cervical cancer lesions, possibly reflecting an ongoing, but ***ineffective*** , ***immune*** ***response*** to the tumour. Recently, evidence has accumulated for systemically impaired T-cell functions in cancer patients, associated with decreased expression of signal-transducing zeta (zeta) chain dimer molecules on circulating T-cells and NK-cells. Here, we report on the intralesional downregulation of zeta chain expression on T-cells in cervical carcinoma. Paraffin-embedded or snap-frozen sections from 24 different cervical cancer specimens were studied. Paraffin-embedded tumour-positive (n = 7) and tumour-negative (n = 15) pelvic lymph nodes were also included in the study. Immunostaining was performed on consecutive sections with antibodies specific for CD3-epsilon or the

CD3-associated zeta chain dimer. Antigen retrieval by sodium citrate/microwave treatment was essential for zeta staining of paraffin sections. The amount of zeta positive cells was quantitated and related to the number of CD3-epsilon+ cells in corresponding tumour areas. Of the 24 cervical cancer specimens studied, zeta chain dimer expression was reduced in seven cases and strongly reduced in the other 17 samples. In tonsil control sections, CD3-epsilon and CD3-zeta were always co-expressed in almost equal numbers. Also, both tumour-negative and -positive lymph nodes showed zeta chain expression which equalled that of CD3-epsilon expression. These data indicate that a decreased expression of signal-transducing zeta molecules on tumour-infiltrating T-cells is frequent in cervical cancer. The apparently unimpaired zeta chain expression within draining lymph nodes suggests that local tumour-derived factors at the primary site are instrumental in zeta chain down-regulation.

L6 ANSWER 27 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 1997:393692 BIOSIS <<LOGINID::20100316>>
DN PREV199799692895
TI The viral envelope in the evolution of HIV: A hypothetical approach to inducing an effective immune response to the virus.
AU Ngu, V. A.
CS Cancer Res. Lab., Fac. Med. Biomed. Sci., B.P. 1364, Yaounde, Cameroon
SO Medical Hypotheses, (1997) Vol. 48, No. 6, pp. 517-521.
CODEN: MEHYDY. ISSN: 0306-9877.
DT Article
LA English
ED Entered STN: 10 Sep 1997
Last Updated on STN: 10 Sep 1997
AB The human immunodeficiency virus (HIV) is 'perceived' by the host immune system as partly-self because of the presence of host cell wall membrane on the viral envelope. This perception leads to an ***ineffective*** ***immune*** ***response*** to the virus. It is proposed that only viral core antigens without the envelope will be perceived as non-self by the host immune system and can provoke an effective immune response. In normal uninfected persons, core antigens could therefore serve as a vaccine. In HIV infected persons, uncommitted immunocytes from the peripheral leucocytes freed from antibodies will in vitro process autologous viral core antigens as non-self antigens and lead to an effective immune response against the HIV when reinjected into the patient. The use of autologous viral core antigens provides, at the same time, a means for testing viral core antigens as possible vaccines without any risk to a third person. This immunotherapy of the HIV, when confirmed, will support core antigens as possible vaccines and could also be applied to the large group of retroviral and other enveloped viruses that cause chronic infections and malignant tumours in man and animals, with considerable benefits to human and animal health.

L6 ANSWER 28 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 1996:562637 BIOSIS <<LOGINID::20100316>>
DN PREV199799291993
TI Molecular analysis of mixed infection with hepatitis C virus and human immunodeficiency virus in a patient infected simultaneously.
AU Mazza, Cinzia; Puoti, Massimo; Ravaggi, Antonella; Castelnuovo, Filippo; Albertini, Alberto; Cariani, Elisabetta [Reprint author]

CS III Lab. Analisi, Spedali Civili, p.le Spedali Civili 1, 25123 Brescia, Italy
SO Journal of Medical Virology, (1996) Vol. 50, No. 3, pp. 276-282. CODEN: JMVIDB. ISSN: 0146-6615.
DT Article
LA English
ED Entered STN: 23 Dec 1996
Last Updated on STN: 23 Dec 1996
AB A case of simultaneous infection with HIV and HCV characterized by a rapidly progressive clinical course was studied retrospectively over 3.5 years. Molecular analysis indicated interference between HIV and HCV and between HCV subtypes 1a and 1b. An ***ineffective*** ***immune*** ***response*** was suggested by the persistence and sequence conservation of the HCV HVR1 variants isolated during the follow-up.

L6 ANSWER 29 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 1996:23489 BIOSIS <<LOGINID::20100316>>
DN PREV199698595624
TI Respiratory syncytial virus induces interleukin-10 by human alveolar macrophages: Suppression of early cytokine production and implications for incomplete immunity.
AU Panuska, James R. [Reprint author]; Merolla, Rocco; Rebert, Nancy A.; Hoffmann, Stephen P.; Tsivitse, Paul; Cirino, Nick M.; Sivlerman, Robert H.; Rankin, John A.
CS Airway Disease Center, 2074 Abington Rd., Dep. Med., Univ. Hospital Case Western Reserve Univ., Cleveland, OH 44106, USA
SO Journal of Clinical Investigation, (1995) Vol. 96, No. 5, pp. 2445-2453. CODEN: JCINAO. ISSN: 0021-9738.
DT Article
LA English
ED Entered STN: 12 Jan 1996
Last Updated on STN: 12 Jan 1996
AB Respiratory syncytial virus (RSV) causes repeated infections thought to be due to an ***ineffective*** ***immune*** ***response*** . We examined the hypothesis that incomplete immunity may result, in part, from RSV-infected alveolar macrophage production of IL-10 which can interfere with the production of immunoregulatory cytokines. We also assessed whether RSV induced the expression of the 2',5' oligoadenylate (2-5A)-dependent RNase L, an endoribonuclease involved in the antiviral activities of interferons. Human alveolar macrophages were exposed to medium (uninfected control), RSV, LPS, and RSV + LPS then were assessed for expression of the cytokines TNF-alpha, IL-1-beta, IL-8, IL-10, as well as 2-5A-dependent RNase L. LPS up-regulated the expression of protein and mRNA for all cytokines. RSV stimulated the protein levels of TNF-alpha, did not alter IL-1-beta, and decreased IL-8. RSV markedly stimulated protein expression of IL-10 and 2-5A-dependent RNase L. RSV had minor effects on the steady state mRNA levels of TNF-alpha, IL-1-beta, and IL-8, yet potently induced IL-10. Cells costimulated with RSV + LPS demonstrated reduced protein and mRNA levels of TNF-alpha, IL-1-beta, IL-8 but synergistically increased IL-10 levels compared to RSV- or LPS-activated cells. Kinetic analysis indicated that RSV induced a delayed and sustained increase in IL-10 transcripts. Furthermore, RSV-infected alveolar macrophage supernatants suppressed IL-1-beta and IL-8 production by LPS-stimulated alveolar macrophages as did recombinant IL-10. Anti-IL-10 neutralized these effects. These studies indicate that RSV is capable of suppressing production of early immunoregulatory

cytokines through induction of IL-10 perhaps mediated by 2-5A-dependent RNase L (or other endoribonucleases) accounting for the ***ineffective*** ***immune*** ***response*** to this virus.

L6 ANSWER 30 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 1995:85318 BIOSIS <<LOGINID::20100316>>
DN PREV199598099618
TI Viral antibody titers are influenced by HLA-DR2 phenotype.
AU Lio, Domenico; Caccamo, Nadia; D'Anna, Claudia; Cigna, Diego; Candore, Giuseppina; Caruso, Calogerro [Reprint author]
CS Ist. Patol. Gen., Univ. Palermo, Corso Tukory 211, I-90134 Palermo, Italy
SO Experimental and Clinical Immunogenetics, (1994) Vol. 11, No. 4, pp. 182-186.
CODEN: ECIME4. ISSN: 0254-9670.
DT Article
LA English
ED Entered STN: 22 Feb 1995
Last Updated on STN: 22 Feb 1995
AB Antibody serum levels against herpes simplex type 1 virus, cytomegalovirus, viral capsid antigens of Epstein-Barr virus, and rubella virus were evaluated in a sample of the Sicilian population. Results demonstrated that HLA-DR2-positive individuals showed a significant increase in antibody titers, when compared to HLA-DR2-negative individuals. These observations seem to be in contrast with the reported association of the HLA-R2 phenotype with an ***ineffective*** ***immune*** ***response*** against several infectious pathogens. On the other hand, an increase humoral response to viral antigens need not be interpreted as a marker for effective control of virus infections. In fact, the response to virus infections is related to the T-cell-mediated immune response restricted by class-I- or class-II-encoded proteins. Thus, the above-mentioned HLA-DR2-related susceptibility to certain viral infections could be associated with a preferential induction of an increased (although ***ineffective***) antibody synthesis against viral antigens.

L6 ANSWER 31 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 1994:438486 BIOSIS <<LOGINID::20100316>>
DN PREV199497451486
TI Immunological parameters in peripheral blood of patients with renal cell carcinoma before and after nephrectomy.
AU Dadian, G. [Reprint author]; Riches, P. G. [Reprint author]; Henderson, D. C. [Reprint author]; Taylor, A.; Moore, J.; Atkinson, H.; Gore, M. E.
CS Dep. Immunol., Chelsea Westminster Hosp., London, UK
SO British Journal of Urology, (1994) Vol. 74, No. 1, pp. 15-22.
CODEN: BJURAN. ISSN: 0007-1331.
DT Article
LA English
ED Entered STN: 11 Oct 1994
Last Updated on STN: 11 Oct 1994
AB Objective: To determine the effects of nephrectomy on the immune response of patients with renal cell carcinoma (RCC). Patients and methods: Five patients with RCC were monitored before and over a period of up to 3 months after nephrectomy. The aspects measured were the phenotypic expression of markers on circulating lymphocytes, circulating concentrations of cytokines. markers of inflammatory and immune responses,

and natural killer (NK) cell and lymphokine-activated killer (LAK) cell activity in peripheral blood mononuclear cells (PBMC). The suppressive activity of patients' plasma on NK activity and ability to generate interleukin-2 (IL-2) induced LAK cells in PBMC of normal volunteers was also investigated. Results: The results indicated that high CD4/8 ratios were present pre-nephrectomy with evidence of inflammatory responses and immune activation in some patients, particularly those with metastatic disease. Conclusion: The effect of nephrectomy was to decrease the inflammatory response and increase immune activation. Various defects in NK cell activity and LAK cell generation were demonstrated pre-surgery which slowly improved once the primary tumour had been removed and it is suggested that such defects could have contributed to tumour growth and development due to an ***ineffective*** ***immune*** ***response*** .

L6 ANSWER 32 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 1991:387352 BIOSIS <<LOGINID::20100316>>
DN PREV199192064667; BA92:64667
TI NEONATAL TETANUS DESPITE PROTECTIVE SERUM ANTITOXIN CONCENTRATION.
AU MASELLE S Y [Reprint author]; MATRE R; MBISE R; HOFSTAD T
CS BROEGELMANN RESEARCH LAB MICROBIOL, ARMAUER HANSEN HOUSE, N-5021 BERGEN, NORWAY
SO FEMS (Federation of European Microbiological Societies) Microbiology Immunology, (1991) Vol. 76, No. 3, pp. 171-176.
ISSN: 0920-8534.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 27 Aug 1991
Last Updated on STN: 27 Aug 1991
AB Using the ELISA technique to estimate serum antibodies against tetanus toxin, seven neonates with clinical tetanus were found to have antibody levels 4-13 times higher than the presumed minimum protective level of 0.01 IU/ml. All but one of their mothers had been vaccinated with tetanus toxoid in pregnancy. In two other neonates, whose mothers had received multiple booster doses of toxoid during pregnancy, the anti-toxin concentrations were 100- and 400-times the presumed protective level. Therefore the toxin dose may overwhelm the pre-existing anti-toxin level and produce disease. Furthermore, multiple booster injections of tetanus toxoid may not only enhance serum anti-toxin titres, but could also lead to an ***ineffective*** ***immune*** ***response*** .

L6 ANSWER 33 OF 68 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 1985:297325 BIOSIS <<LOGINID::20100316>>
DN PREV198579077321; BA79:77321
TI MONOCYTE FUNCTION IN THE ACQUIRED IMMUNE DEFICIENCY SYNDROME DEFECTIVE CHEMOTAXIS.
AU SMITH P D [Reprint author]; OHURA K; MASUR H; LANE H C; FAUCI A S; WAHL S M
CS LAB MICROBIOL IMMUNOL, NATIONAL INST DENTAL RES, NATIONAL INST HEALTH, BETHESDA, MD 20205, USA
SO Journal of Clinical Investigation, (1984) Vol. 74, No. 6, pp. 2121-2128.
CODEN: JCINAO. ISSN: 0021-9738.
DT Article
FS BA

LA ENGLISH

AB The ***ineffective*** ***immune*** ***response*** in patients with the acquired immune deficiency syndrome (AIDS) contributes to severe and widespread infections and unrestricted growth by certain tumors. To determine whether monocyte dysfunction contributes to this immunosuppressed condition, monocyte chemotaxis was investigated in patients with AIDS. Using 3 different chemotactic stimuli, N-formylmethionylleucylphenylalanine, lymphocyte-derived chemotactic factor and C5a des Arg, the chemotactic responses of monocytes from 7 homosexual men with AIDS, 3 homosexuals with lymphadenopathy and an abnormal immunological profile, 7 healthy homosexual men and 23 heterosexual control individuals were studied. Monocytes from each of the AIDS patients with Kaposi's sarcoma and/or opportunistic infection exhibited a marked reduction in chemotaxis to all stimuli compared with the healthy control subjects. The reduced chemotactic responses were observed over a wide range of concentrations for each stimulus. Monocytes from AIDS patients who had clinically apparent opportunistic infection(s) exhibited a greater reduction in monocyte migration to all 3 stimuli than monocytes from the AIDS patients with only Kaposi's sarcoma. Monocytes from each of 3 homosexuals with lymphadenopathy and an abnormal immunological profile exhibited decreased chemotactic responses that were intermediate between those of the AIDS patients and the healthy heterosexual control subjects. In contrast to these findings, monocytes from each of 7 healthy homosexuals exhibited normal chemotactic responses to the same stimuli. In addition, monocytes from AIDS patients exhibited reduced chemotaxis to soluble products of Giardia lamblia, 1 of several protozoan parasites prevalent in AIDS patients. Thus, the immune abnormality in AIDS, previously thought to involve only the T-, B- and natural killer lymphocytes, extends to the monocyte-macrophage. Defective monocyte migratory function may contribute to the depressed inflammatory response to certain organisms and to the apparent unrestricted growth of certain neoplasms in patients with AIDS.

L6 ANSWER 34 OF 68 CAPLUS COPYRIGHT 2010 ACS on STN

AN 2010:133213 CAPLUS <<LOGINID::20100316>>

TI Hepatitis B virus overexpresses suppressor of cytokine signaling-3 (SOCS3) thereby contributing to severity of inflammation in the liver

AU Koeberlein, Bernd; zur Hausen, Axel; Bektas, Nuran; Zentgraf, Hanswalter; Chin, Ruth; Toan, Nguyen Linh; Kandolf, Reinhard; Torresi, Joseph; Bock, C.-Thomas

CS Department of Molecular Pathology, Institute of Pathology, University Hospital of Tuebingen, Tuebingen, 72076, Germany

SO Virus Research (2010), 148(1-2), 51-59
CODEN: VIREDF; ISSN: 0168-1702

PB Elsevier B.V.

DT Journal

LA English

AB The mechanism by which hepatitis B virus (HBV) infection causes severe inflammatory liver diseases is multifactorial and related to interactions with cell signaling pathways and the ensuing inflammatory response. Activation of JAK/STAT/SOCS signaling is essential for the induction of cellular antiviral responses, contributes to apoptosis and is neg. regulated by SOCS proteins. Recent reports have shown that SOCS3 activation interferes with viral protein expression and treatment response and thereby plays a major role in hepatitis virus infections. We analyzed the expression of SOCS3 in liver specimens from HBV-infected patients using immunohistochem. (IHC) and detd. the effect of HBV on STAT/SOCS

signaling in functional cell culture expts. (HuH-7) using HBV-expressing adenoviral constructs (AdHBV). Increased expression of SOCS3 protein was identified in liver specimens from patients with chronic HBV-infection and this correlated with the severity of liver inflammation. In accordance with the IHC-findings, in vitro analyses demonstrated that HBV infection of HuH7 cells was assocd. with increased expression of SOCS3 protein. In spite of the over expression of its neg. regulator SOCS3 we obsd. a constitutive activation of STAT3. SOCS1 levels were not increased while pSTAT1 was suppressed in HBV-infected HuH7 cells. Our results demonstrate that STAT/SOCS-signaling is dysregulated in HBV-infected hepatocytes both in vivo and in vitro and this correlated with the severity of liver inflammatory changes. This interference of STAT/SOCS signaling by HBV may result in an ***ineffective*** ***immune*** ***response*** against HBV and potentially contributes to viral pathogenesis, malignant transformation and may represent an important mechanism of viral persistence.

L6 ANSWER 35 OF 68 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2006:684086 CAPLUS <<LOGINID::20100316>>
DN 145:333683
TI Human leukocyte antigen distribution analysis in North Italian brain Glioma patients: an association with HLA-DRB1*14
AU Guerini, Franca R.; Agliardi, Cristina; Zanzottera, Milena; Delbue, Serena; Pagani, Elisabetta; Tinelli, Carmine; Boldorini, Renzo; Car, Pier Giorgio; Veggiani, Claudia; Ferrante, Pasquale
CS Laboratory of Molecular Medicine and Biotechnology, S. Maria Nascente, Don C. Gnocchi Foundation IRCCS, Milan, Italy
SO Journal of Neuro-Oncology (2006), 77(2), 213-217
CODEN: JNODD2; ISSN: 0167-594X
PB Springer
DT Journal
LA English
AB Human leukocyte antigens (HLA) are widely expressed cell surface mols. that present antigenic peptides to T-lymphocytes and modulate the immune response against inflammatory and malignant disease. Frequently, tumoral cells express antigens that are recognized by the immune system. ***Ineffective*** ***immune*** ***response*** could be the result of defects in antigen presentation in those subjects with peculiar HLA alleles, which, owing to mechanisms that are still unknown, are unable to carry out their function. Only a few studies on glioma and HLA assocn. have been performed to date. The aim of our study was to characterize a group of Italian Caucasian patients with glioma, to investigate a possible assocn. between HLA antigens and cerebral glioma tumorigenesis in Italian patients. HLA typing of class I and class II loci was done by mol. typing performed on blood DNA from 36 glioma patients from northern Italy. The data obtained were compared with HLA frequencies taken from the database of northern Italian organ donors. A pos. assocn. between HLA-DRB1*14 and the presence of symptomatic cerebral glioma was obsd. (p = 0.02, odds ratio = 2.48, 95% confidence interval: 1.09-5.45). This is the first Italian report on a case-control data study of HLA distribution conducted on a group of glioma patients and a first step in defining a possible involvement of HLA in susceptibility to brain glioma in the Italian population.
OSC.G 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 36 OF 68 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2006:464136 CAPLUS <<LOGINID::20100316>>
DN 145:374886
TI Efficient Presentation of Naturally Processed HLA Class I Peptides by Artificial Antigen-Presenting Cells for the Generation of Effective Antitumor Responses
AU Hirano, Naoto; Butler, Marcus O.; Xia, Zhinan; Berezovskaya, Alla; Murray, Andrew P.; Ansen, Sascha; Nadler, Lee M.
CS Department of Medical Oncology, Dana Farber Cancer Institute, Boston, MA, 02115, USA
SO Clinical Cancer Research (2006), 12(10), 2967-2975
CODEN: CCREF4; ISSN: 1078-0432
PB American Association for Cancer Research
DT Journal
LA English
AB Appropriate presentation of tumor-assocd. antigens (TAA) by antigen-presenting cells (APC) is required for the development of clin. relevant antitumor T-cell responses. One common approach, which uses APC pulsed with synthetic peptides, can sometimes generate ***ineffective*** ***immune*** ***responses*** . This failure may, in part, be attributed to the formation of HLA/synthetic pulsed peptide complexes that possess different conformations compared with those of endogenously presented peptides. In addn., endogenous peptides may undergo post-translational modifications, which do not occur with synthetic peptides. Because their goal is to induce immunity that can recognize TAA that are endogenously presented by tumors, the authors designed an APC that would not only express the required immunoaccessory mols. but also naturally process and present target antigenic peptides. Here, the authors generated an artificial APC (aAPC) that can endogenously present any chosen HLA-A*0201 (A2)-restricted peptide by processing a fusion protein that contains a unique "LTK" sequence linked to the antigenic peptide. Proteasome-dependent processing is so effective that the presented peptide can be directly eluted from the cell surface and identified by biochem. methods. Furthermore, the authors found that aAPC, engineered to endogenously present peptide derived from the melanoma antigen MART1, can be used to prime and expand antitumor CTL that target MART1-expressing tumor cells in a HLA-A2-restricted manner. The authors' engineered aAPC could serve as an "off-the-shelf" APC designed to constitutively express class I-restricted TAA peptides and could be used to generate effective T-cell responses to treat human disease.
OSC.G 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)
RE.CNT 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 37 OF 68 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2005:481243 CAPLUS <<LOGINID::20100316>>
DN 143:210044
TI Immune responses following mycoplasma infection
AU Simecka, Jerry W.
CS Department of Molecular Biology and Immunology, University of North Texas Health Science Center, Fort Worth, TX, 76107, USA
SO Mycoplasmas (2005), 485-534. Editor(s): Blanchard, Alain; Browning, Glenn. Publisher: Horizon Bioscience, Wymondham, UK.
CODEN: 69GWJ6; ISBN: 0-8493-9861-4
DT Conference; General Review
LA English
AB A review. Mycoplasmas are responsible for many human and animal

respiratory diseases and have a tremendous economic and health impact worldwide. Because of the chronic nature of these infections, it is likely that almost every component of the host immune system is involved in the response to mycoplasma disease. Innate immunity is crit. in the early clearance and control of infection. Adaptive immune responses against mycoplasma have contrasting impacts on infection and the pathogenesis of infection. Vaccines can induce immune responses that resist infection. In addn., immune responses also minimize the potential spread of infection to other tissues, which can lead to arthritis and other diseases. However, the hallmark of many mycoplasma diseases is the persistence of the organism, and the provocation of frustrated and ***ineffective*** ***immune*** ***responses*** against the infection results in the development of chronic inflammation. Despite the vast amt. of research, the mechanisms that control hosts' resistance and susceptibility to mycoplasma infection remains unclear. Immune responses are believed to be crit. players in the pathogenesis of mycoplasma disease. In this review, we will highlight the potential roles of innate and adaptive immunity as influential mediators in animal and human mycoplasma pathogenesis and resistance infection.

OSC.G 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)
RE.CNT 261 THERE ARE 261 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 38 OF 68 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2005:395589 CAPLUS <<LOGINID::20100316>>
DN 142:423905
TI Method of therapy of disease based on immune cycling and presence of regulator cells
IN Ashdown, Martin Leonard
PA Immunaid Pty. Ltd., Australia
SO PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005040816	A1	20050506	WO 2004-AU1456	20041022
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2004283322	A1	20050506	AU 2004-283322	20041022
	CA 2543490	A1	20050506	CA 2004-2543490	20041022
	EP 1692516	A1	20060823	EP 2004-761461	20041022
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				
	BR 2004015533	A	20061226	BR 2004-15533	20041022
	CN 1898569	A	20070117	CN 2004-80038999	20041022
	JP 2007509078	T	20070412	JP 2006-535913	20041022

	MX 2006004522	A	20061110	MX 2006-4522	20060424
	US 20070202119	A1	20070830	US 2007-576981	20070302
PRAI	AU 2003-905858	A	20031024		
	WO 2004-AU1456	W	20041022		

AB The present inventor has surprisingly found that the immune system is cycling during disease states characterized by the presence of regulator cells. While not wishing to be limited by theory, it appears that effector cell expansion against a target antigen is followed by the expansion of regulator cells directed against the effectors. Upon control of the effector cells by the regulator cells the nos. and/or activity of both types of cells decrease, which in turn is followed by the same cycle due to the continuous presence or incomplete removal of antigen which results in an oscillating persistent, but ***ineffective*** , ***immune*** ***response*** against the, for example, tumor or virus. The present invention provides a method of treating a disease characterized by the prodn. of regulator cells, the method comprising; (i) monitoring a patient suffering from the disease for at least one of: (a) no. and/or activity of regulator cells, (b) no. and/or activity of effector cells, (c) a mol. assocd. with the disease, and/or (d) an immune system marker, and (ii) exposing the patient to an agent to treat the disease, wherein the timing of administration of the agent is selected such that the activity of the effector cells is not significantly reduced. Based on these observations, the present invention provides methods for treating diseases such as cancer and a HIV infection.

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 39 OF 68 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2003:542785 CAPLUS <<LOGINID::20100316>>
DN 139:115941
TI Dynamics of cytokine generation in patients with active pulmonary tuberculosis
AU Jo, Eun-Kyeong; Park, Jeong-Kyu; Dockrell, Hazel M.
CS Department of Microbiology, College of Medicine, Chungnam National University, Daejeon, S. Korea
SO Current Opinion in Infectious Diseases (2003), 16(3), 205-210
CODEN: COIDE5; ISSN: 0951-7375
PB Lippincott Williams & Wilkins
DT Journal; General Review
LA English

AB A review. Cytokines have been implicated in the protective immunity, pathophysiol., and development of tuberculosis. Most people who become infected with Mycobacterium tuberculosis mount an effective protective immune response, but 5-10% develop disease. Active pulmonary tuberculosis can be considered to reflect an ***ineffective*** ***immune*** ***response*** against mycobacterial infection. A better understanding of how cytokine prodn. contributes to immunity and pathol. would aid the development of new vaccines and therapeutic strategies. At the time of diagnosis, prodn. of M. tuberculosis or mycobacterial antigen-induced interferon-.gamma. by peripheral blood mononuclear cells from tuberculosis patients is usually depressed, compared with that of healthy control subjects, whereas cytokine prodn. at the site of disease is elevated. In most patients, depressed interferon-.gamma. prodn. by peripheral blood mononuclear cells seems to be a transient response because it is increased in most active tuberculosis patients during and following successful antituberculous therapy. However, some patients remain anergic in vivo and in vitro after chemotherapy. Among the cytokines contributing to

protective immunity, interleukins 12 and 18, and tumor necrosis factor-.alpha. are important.
OSC.G 31 THERE ARE 31 CAPLUS RECORDS THAT CITE THIS RECORD (31 CITINGS)
RE.CNT 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 40 OF 68 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2002:400294 CAPLUS <<LOGINID::20100316>>
DN 137:246109
TI RSV-induced immunopathology: Dynamic interplay between the virus and host immune response
AU Varga, Steven M.; Braciale, Thomas J.
CS University of Virginia Health Sciences Center, Beirne B. Carter Center for Immunology Research, Charlottesville, VA, 22908, USA
SO Virology (2002), 295(2), 203-207
CODEN: VIRLAX; ISSN: 0042-6822
PB Elsevier Science
DT Journal; General Review
LA English
AB A review on the role of the respiratory syncytial virus (RSV) in eliciting an ***ineffective*** ***immune*** ***response*** in humans that allows for repeated infections throughout life. There is currently no safe and effective licensed RSV vaccine in part because the mechanism behind the formalin-inactivated RSV vaccine-enhanced disease remains poorly understood. Thus, a better understanding of both the primary and the secondary immune response to RSV is imperative before a safe and successful vaccine can be developed.
OSC.G 24 THERE ARE 24 CAPLUS RECORDS THAT CITE THIS RECORD (24 CITINGS)
RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 41 OF 68 CAPLUS COPYRIGHT 2010 ACS on STN
AN 1997:411547 CAPLUS <<LOGINID::20100316>>
DN 127:147595
OREF 127:28477a
TI Hodgkin's disease: a tumor with disturbed immunological pathways
AU Gruss, Hans-Jurgen; Pinto, Antonio; Duyster, Justus; Poppema, Sibrand; Herrmann, Friedhelm
CS Dep. Hematology Oncology, Clinical Immunology and Infectious Disease, Univ. Ulm, Ulm, D-89081, Germany
SO Immunology Today (1997), 18(4), 156-163
CODEN: IMTOD8; ISSN: 0167-4919
PB Elsevier
DT Journal; General Review
LA English
AB A review with 73 refs. Hodgkin's disease is a lymphoid neoplasia characterized by a low frequency of malignant Hodgkin and Reed-Sternberg (H-RS) cells in an abundant background of non-neoplastic cells. H-RS cells and their neighbors interact via a complex network of cellular activation/adhesion mols. and cytokines. Here, the authors suggest that H-RS cells can be regarded as antigen-presenting cells able to interact with surrounding T cells, resulting in an intense, but ***ineffective*** , ***immune*** ***response*** .
OSC.G 78 THERE ARE 78 CAPLUS RECORDS THAT CITE THIS RECORD (78 CITINGS)

L6 ANSWER 42 OF 68 CAPLUS COPYRIGHT 2010 ACS on STN
AN 1997:209360 CAPLUS <<LOGINID::20100316>>

DN 126:275982
OREF 126:53488h,53489a
TI Melanoma vaccines: prospects for the treatment of melanoma
AU Hersey, Peter
CS Oncol. & Immunol. Unit, Dep. of Surg., John Hunter Hosp., Newcastle, 2300, Australia
SO Expert Opinion on Investigational Drugs (1997), 6(3), 267-278
CODEN: EOIDER; ISSN: 0967-8298
PB Ashley Publications
DT Journal; General Review
LA English
AB A review with 95 refs. A no. of melanoma vaccines, made from whole melanoma cells or components of melanoma cells, are being tested in Phase II or III trials in patients after surgical removal of high risk primary or regional lymph node metastases, or in those with disseminated melanoma. During the progress of these trials, a no. of melanoma antigens and their peptide epitopes that are recognized by human T-cells have been described. These findings and new information about antigen recognition by human T-cells have made it possible to explore the use of peptide epitopes targeted at T-cells as melanoma vaccines. Preliminary results are encouraging and suggest that it may soon be possible to use well defined vaccines, selected on the basis of the antigenic phenotype of the patient's melanoma and their HLA status. Equally exciting advances have been made prepg. and using recombinant viral vectors contg. genes that code for melanoma antigens. Exptl. studies on the use of naked DNA as vaccines are also proceeding. Several fundamental obstacles preventing the effective use of T-cell epitope vaccines remain. These include selection of HLA and tumor antigen loss variants by the immune system, and conditioning of an ***ineffective*** ***immune*** ***response*** by the growing tumor. These aspects suggest that the development of effective vaccine therapy in the future may require a combination of strategies designed to stimulate HLA-restricted and -non-restricted effector cells, and judicious use of cytokines to obtain an effective immune response.
OSC.G 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)

L6 ANSWER 43 OF 68 CAPLUS COPYRIGHT 2010 ACS on STN
AN 1996:235582 CAPLUS <<LOGINID::20100316>>
DN 124:314372
OREF 124:58282h,58283a
TI Hepatitis B virus immunopathology
AU Chisari, Francis V.; Ferrari, Carlo
CS Department Molecular and Experimental Medicine, Scripps Research Institute, La Jolla, CA, 92037, USA
SO Springer Seminars in Immunopathology (1995), 17(2/3), 261-81
CODEN: SSIMDV; ISSN: 0344-4325
PB Springer
DT Journal; General Review
LA English
AB A review, with 83 refs. Approx. 5% of the world population is infected by the hepatitis B virus (HBV) which causes a necroinflammatory liver disease of variable duration and severity. Chronically infected patients with active liver disease carry a high risk of developing cirrhosis and hepatocellular carcinoma. The immune response to HBV-encoded antigens is responsible both for viral clearance and for disease pathogenesis during this infection. While the humoral antibody response to viral envelope antigens contributes to the clearance of circulating virus particles, the

cellular immune response to the envelope, nucleocapsid and polymerase antigens eliminates infected cells. The class I- and class II-restricted T cell responses to the virus are vigorous, polyclonal and multispecific in acutely infected patients who successfully clear the virus, and they are relatively weak and more narrowly focussed in chronically infected patients who do not. The pathogenetic and antiviral potential of the cytotoxic T lymphocyte (CTL) response to HBV have been demonstrated by the induction of a severe necroinflammatory liver disease following the adoptive transfer of HBV surface antigen-specific CTL into HBV transgenic mice, and by the noncytolytic suppression of viral gene expression and replication in the same animals by a post-transcriptional mechanism mediated by interferon-.gamma., tumor necrosis factor-.alpha. and interleukin-2. The dominant cause of viral persistence during HBV infection is the development of a weak antiviral immune response to the viral antigens. While neonatal tolerance probably plays an important role in viral persistence in patients infected at birth, the basis for poor responsiveness in adult onset infection is not well understood and requires further anal. Viral evasion by epitope inactivation and T cell receptor antagonism may contribute to the worsening of viral persistence in the setting of an ***ineffective*** ***immune*** ***response*** , as can the incomplete down-regulation of viral gene expression and the infection of immunol. privileged tissues. Chronic liver cell injury and the attendant inflammatory and regenerative responses create the mutagenic and mitogenic stimuli for the development of DNA damage that can cause hepatocellular carcinoma.

OSC.G 68 THERE ARE 68 CAPLUS RECORDS THAT CITE THIS RECORD (68 CITINGS)

L6 ANSWER 44 OF 68 CAPLUS COPYRIGHT 2010 ACS on STN
AN 1995:512568 CAPLUS <<LOGINID::20100316>>
DN 122:263015
OREF 122:47993a,47996a
TI Hepatitis B virus immunopathogenesis
AU Chisari, Francis V.; Ferrari, Carlo
CS Dep. Mol. Experimental Med., Scripps Res. Inst., La Jolla, CA, 92037, USA
SO Annual Review of Immunology (1995), 13, 29-60
CODEN: ARIMDU; ISSN: 0732-0582
PB Annual Reviews
DT Journal; General Review
LA English
AB A review with 116 refs. Approx. 5% of the world population is infected by the hepatitis B virus (HBV) that causes a necroinflammatory liver disease of variable duration and severity. Chronically infected patients with active liver disease carry a high risk of developing cirrhosis and hepatocellular carcinoma. The immune response to HBV-encoded antigens is responsible both for viral clearance and for disease pathogenesis during this infection. While the humoral antibody response to viral envelope antigens contributes to the clearance of circulating virus particles, the cellular immune response to the envelope, nucleocapsid, and polymerase antigens eliminates infected cells. The class I- and class II-restricted T cell responses to the virus are vigorous, polyclonal, and multispecific in acutely infected patients who successfully clear the virus, and the responses are relatively weak and more narrowly focused in chronically infected patients who do not. The pathogenetic and antiviral potential of the cytotoxic T lymphocyte (CTL) response to HBV has been demonstrated by the induction of a severe necroinflammatory liver disease following the adoptive transfer of HBsAg-specific CTL into HBV transgenic mice, and by the noncytolytic suppression of viral gene expression and replication in

the same animals by a posttranscriptional mechanism mediated by interferon gamma, tumor necrosis factor alpha, and interleukin 2. The dominant cause of viral persistence during HBV infection is the development of a weak antiviral immune response to the viral antigens. While neonatal tolerance probably plays an important role in viral persistence in patients infected at birth, the basis for poor responsiveness in adult-onset infection is not well understood and requires further anal. Viral evasion by epitope inactivation and T cell receptor antagonism may contribute to the worsening of viral persistence in the setting of an ***ineffective*** ***immune*** ***response*** , as can the incomplete downregulation of viral gene expression and the infection of immunol. privileged tissues. Chronic liver cell injury and the attendant inflammatory and regenerative responses create the mutagenic and mitogenic stimuli for the development of DNA damage that can cause hepatocellular carcinoma. Elucidation of the immunol. and virol. basis for HBV persistence may yield immunotherapeutic and antiviral strategies to terminate chronic HBV infection and reduce the risk of its life-threatening sequelae.

OSC.G 750 THERE ARE 750 CAPLUS RECORDS THAT CITE THIS RECORD (751 CITINGS)

L6 ANSWER 45 OF 68 CAPLUS COPYRIGHT 2010 ACS on STN
AN 1973:66957 CAPLUS <<LOGINID::20100316>>
DN 78:66957
OREF 78:10571a,10574a
TI Penicillin treatment and antibody response of pigs experimentally infected with Erysipelothrix insidiosa
AU Azechi, Hayami; Terakado, Nobuyuki; Ninomiya, Kiyoji
CS Natl. Vet. Assay Lab., Tokyo, Japan
SO American Journal of Veterinary Research (1972), 33(10), 1963-73
CODEN: AJVRAH; ISSN: 0002-9645
DT Journal
LA English
AB Procaine penicillin G [6130-64-9] (50,000 units/kg/day, i.m., for 3 days) had a complete chemotherapeutic effect in pigs exptl. infected with Erysipelothrix insidiosa 62 hr earlier. Serum antibodies were detected in infected pigs after this dose regimen of penicillin, but were not detected in pigs in which the penicillin treatment began 2 hr after they were infected. The time after infection of penicillin administration appeared to be an important factor in serum antibody prodn. The ***ineffective*** ***immune*** ***response*** of pigs treated with penicillin in the early stage of infection might be due to the inhibition of bacterial multiplication caused by the drug present in the treated pigs. Accordingly, if a given quant. of bacilli is allowed to persist or multiply in the pig for at least 12 hr before it is treated with penicillin, the pig (in the early phase of fever) will acquire immunity against reinfection with E. insidiosa.

L6 ANSWER 46 OF 68 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 2006553977 EMBASE <<LOGINID::20100316>>
TI [Hemophagocytic syndrome].
Hamophagozytische syndrome.
AU Janka, G., Dr. Prof. (correspondence)
CS Zentrum fur Frauenheilkunde, Kinder- und Jugendmedizin, Universitatskrankenhaus Hamburg-Eppendorf, Martinistrasse 20, 20246 Hamburg, Germany. janka@uke.uni-hamburg.de
SO Monatsschrift fur Kinderheilkunde, (Nov 2006) Vol. 154, No. 11, pp.

1104-1109.
Refs: 28
ISSN: 0026-9298 CODEN: MOKIAY
CY Germany
DT Journal; Article
FS 025 Hematology
037 Drug Literature Index
LA German
SL English; German
ED Entered STN: 28 Nov 2006
Last Updated on STN: 28 Nov 2006
AB The common basis for hemophagocytic syndromes are genetic or acquired immune defects which lead to an exaggerated but ***ineffective*** ***immune*** ***response*** with high levels of inflammatory cytokines. Cardinal symptoms are prolonged, antibiotic resistant fever, hepatosplenomegaly and bicytopenia or pancytopenia. Hemophagocytosis, which gave hemophagocytic syndrome its name, often develops later during the course of the disease. Characteristic laboratory values are high concentrations of ferritin, triglycerides, lactate dehydrogenase, bilirubin, and transaminases, and low levels of fibrinogen, or a generalized coagulation disorder. In contrast to a normal infection, the symptoms are more pronounced and, most important, they are progressive. Timely treatment with immunomodulatory and cytostatic drugs can suppress hyperinflammation and thus be life-saving. .COPYRGT. 2006 Springer Medizin Verlag.

L6 ANSWER 47 OF 68 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 2003514351 EMBASE <<LOGINID::20100316>>
TI Underlying chronic granulomatous disease in a patient with bronchocentric granulomatosis.
AU Moltyaner, Y.; Geerts, W.H., Dr. (correspondence); Downey, G.P.
CS Division of Respirology, University of Toronto, Toronto, Ont., Canada. william.geerts@swchsc.on.ca
AU Chamberlain, D.W.
CS Department of Pathology, University of Toronto, Toronto, Ont., Canada.
AU Doyle, J.J.
CS Division of Hematology, Hospital for Sick Children, Toronto, Ont., Canada.
AU Heyworth, P.G.
CS Dept. of Molec. and Exp. Medicine, Scripps Research Institute, San Diego, CA, United States.
AU Noack, D.; Rae, J.
CS Department of Immunology, Genentech Inc., South San Francisco, CA, United States.
AU Geerts, W.H., Dr. (correspondence)
CS Sunnybrook and Women's College, Health Sciences Center, 2075 Bayview Avenue, Toronto, Ont. M4N 3M5, Canada. william.geerts@swchsc.on.ca
SO Thorax, (Dec 2003) Vol. 58, No. 12, pp. 1096-1098.
Refs: 17
ISSN: 0040-6376 CODEN: THORA7
CY United Kingdom
DT Journal; Article
FS 015 Chest Diseases, Thoracic Surgery and Tuberculosis
026 Immunology, Serology and Transplantation
037 Drug Literature Index
004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
LA English

SL English
ED Entered STN: 5 Jan 2004
Last Updated on STN: 5 Jan 2004
AB We present a case of bronchocentric granulomatosis in a woman with no history of asthma who was colonised with Aspergillus fumigatus. A family history of chronic granulomatous disease prompted further testing that demonstrated severely depressed neutrophil oxidant production and gp91 phox deficiency compatible with the X linked carrier state of chronic granulomatous disease. Only one report of the association of these two rare diseases has previously appeared in the literature. We postulate that an ***ineffective*** ***immune*** ***response*** led to the prolonged colonisation of A fumigatus resulting in a hypersensitivity reaction that was manifest clinically as bronchocentric granulomatosis.

L6 ANSWER 48 OF 68 EMBASE
AN 2003349692 EMBASE <<LOGINID::20100316>>
TI Myxofibrosarcomas contain large numbers of infiltrating immature dendritic cells.
AU Soilleux, Elizabeth J., Dr. (correspondence); Morris, Lesley S.; Coleman, Nicholas
CS Med. Res. Council Cancer Cell Unit, Hutchison/MRC Research Centre, Cambridge CB2 2XY, United Kingdom.
AU Soilleux, Elizabeth J., Dr. (correspondence); Rous, Brian; Love, Karl; Coleman, Nicholas
CS Department of Histopathology, Addenbrooke's Hospital, Cambridge, United Kingdom.
AU Vowler, Sarah
CS Cambridge University, Ctr. for Applied Medical Statistics, Institute of Public Health, Robinson Way, Cambridge, United Kingdom.
AU Fisher, Cyril
CS Department of Histopathology, Royal Marsden Hospital, London, United Kingdom.
SO American Journal of Clinical Pathology, (1 Apr 2003) Vol. 119, No. 4, pp. 540-545.
Refs: 20
ISSN: 0002-9173 CODEN: AJCPAI
CY United States
DT Journal; Article
FS 016 Cancer
005 General Pathology and Pathological Anatomy
LA English
SL English
ED Entered STN: 18 Sep 2003
Last Updated on STN: 18 Sep 2003
AB Myxofibrosarcoma is a malignant tumor with distinctive histologic features and is believed to be derived from fibroblasts. The function of infiltrating myeloid cells in myxofibrosarcoma is poorly understood. It previously has been shown that a combination of dendritic morphologic features and expression of the C-type lectin, dendritic cell-specific intercellular adhesion molecule-3 grabbing nonintegrin (DC-SIGN), is useful for identifying DC populations in tissue sections. In the present study, we found that 3% to 61% (median, 22)%) of cells in myxofibrosarcomas express DC-SIGN and have dendritic morphologic features. These DC-SIGN-positive cells are not in cell cycle and are consistent with infiltrating DCs. The percentage of DCs in myxofibrosarcomas is independent of tumor grade. It previously has been shown that

DC-SIGN-positive cells are either immature DCs or DCs that predominantly activate TH2 cells, both subsets likely to give rise to ***ineffective*** antitumor responses. The DC-SIGN-positive DCs that we have identified in myxofibrosarcoma may, therefore, be involved in the induction of ***ineffective*** ***immune*** ***responses*** or even tolerance to tumor antigens.

L6 ANSWER 49 OF 68 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 2002424360 EMBASE <<LOGINID::20100316>>
TI Epidemiology and etiology of Hodgkin's lymphoma.
AU Thomas, R.K. (correspondence); Re, D.; Zander, T.; Wolf, J.; Diehl, V.
CS Department of Internal Medicine I, University of Cologne, Cologne, Germany.
SO Annals of Oncology, (2002) Vol. 13, No. SUPPL. 4, pp. 147-152.
Refs: 75
ISSN: 0923-7534 CODEN: ANONE2
CY United Kingdom
DT Journal; Article
FS 016 Cancer
017 Public Health, Social Medicine and Epidemiology
022 Human Genetics
025 Hematology
005 General Pathology and Pathological Anatomy
LA English
SL English
ED Entered STN: 5 Dec 2002
Last Updated on STN: 5 Dec 2002
AB Although scientists have learned much about the derivation of HRS cells, little is known about the basic mechanisms that underlie malignant transformation of their precursors. The HRS cells in cHL have been shown to be derived from preapoptotic germinal center B cells in the majority of cases, while in some cases they seem to be of T-cell origin. The expression of EBV latent genes in EBV positive cases (50%) may be involved in transformation by up-regulation of the transcription factor NF.kappa.B. The transformation process in the EBV negative cases, however, is still not understood. Several studies have focused on the apoptosis resistant phenotype of HRS cells and recent data suggest that constitutively expressed c-FLIP may contribute to apoptosis resistance in cHL. Genetic instability is a typical feature of HRS cells and recent studies point to distinct genetic imbalances rather than subtle genetic alterations such as point mutations or microsatellite instability. Finally, the discovery that the HRS cells themselves contribute to the ***ineffective*** ***immune*** ***response*** by expressing immunosuppressive cytokines or by expressing chemokines that predominantly attract Th2 cells that are unable to kill, has widened our understanding of the environmental crosstalk of these peculiar cells.

L6 ANSWER 50 OF 68 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 2000075117 EMBASE <<LOGINID::20100316>>
TI About some immunologic and immunotherapeutic possibilities in breast cancer.
AU Dimitrov, G. (correspondence); Yovtchev, Y.
CS Department of Surgical Diseases, Medical Faculty, Thracian University, Stara Zagora, Bulgaria.

SO Bulgarian Medicine, (1999) Vol. 7, No. 5-6, pp. 55-56.
Refs: 12
ISSN: 0861-9883 CODEN: BUMEEP
CY Bulgaria
DT Journal; Article
FS 016 Cancer
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LA Bulgarian
SL English; Bulgarian
ED Entered STN: 9 Mar 2000
Last Updated on STN: 9 Mar 2000
AB The immune system proved to be unable of controlling tumor growth patients with cancer. Determining tumor antigenes and identifying ***ineffective*** ***immune*** ***response*** gave possibility the immune system to be activated and manipulating towards destroying of the tumor. The technology of producing and application of monoclonal antibodies became a promissing form of treatment. Fifty percent inhibition of the tumor was achieved with the application of anti-p53 and anti-uPA monoclonal antibodies in patients with breast cancer. These can proved to be important and valuable agents for immuno- and anti-metastatic therapy.

L6 ANSWER 51 OF 68 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 1998392737 EMBASE <<LOGINID::20100316>>
TI The immunopathogenesis of HBV infection.
AU Koziel, M.J. (correspondence)
CS Harvard Institute of Medicine, Beth Israel Deaconness Medical Ctr., Boston, MA, United States. mkoziel@west.bidmc.harvard.edu
SO Antiviral Therapy, (1998) Vol. 3, No. SUPPL. 3, pp. 13-24.
Refs: 101
ISSN: 1359-6535 CODEN: ANTHFA
CY United Kingdom
DT Journal; Conference Article; (Conference paper)
FS 026 Immunology, Serology and Transplantation
037 Drug Literature Index
038 Adverse Reactions Titles
004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
LA English
SL English
ED Entered STN: 10 Dec 1998
Last Updated on STN: 10 Dec 1998
AB Clinical manifestations of hepatitis B virus (HBV) infection are a balance between viral and host factors. The immune response against any virus consists of a coordinated defence of innate immunity and acquired, virus-specific immunity. In acute HBV, immune responses associated with recovery include vigorous, polyclonal CD4 T cells directed against multiple epitopes within HBV; antibodies directed againstsurface envelope proteins (anti-HBs), the development of which requires the presence of a CD4 response; and HBV-specific cytotoxic T lymphocytes (CTLs). HBV-specific CTLs can induce death of infected hepatocytes as well as produce cytokines. Most individuals with acute HBV recover without evidence of massive liver destruction; this, plus evidence from transgenic animal models, suggests that these cytokines produced by T cells play an important role in controlling HBV replication. Individuals who fail to mount a vigorous response in acute HBV develop chronic infection. In

these cases, the persisting ***ineffective*** ***immune*** ***response*** appears to be responsible for liver damage and is likely to initiate the process of hepatic fibrosis. Based on our current understanding of the immune response in acute and chronic HBV, several groups are investigating the prospect of manipulating the immune response in chronic HBV.

L6 ANSWER 52 OF 68 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 1997192505 EMBASE <<LOGINID::20100316>>
TI [Cytokines and HIV infection. Pathophysiology, diagnosis and therapeutic strategies].
Cytokines et infection par le virus de l'immunodeficience humaine (VIH-1): Implications physiopathologiques et consequences diagnostiques et therapeutiques.
AU Guenounou, M. (correspondence)
AU Guenounou, M. (correspondence)
CS Lab. Immunologie/Biologie Cytokines, Centre des Biomolecules, Universite de Reims, 51, Rue Cognacq-Jay, 51096 Reims Cedex 01, France.
SO Immuno-Analyse et Biologie Specialisee, (May 1997) Vol. 12, No. 2, pp. 65-69.
Refs: 50
ISSN: 0923-2532 CODEN: IBSPEW
CY France
DT Journal; General Review; (Review)
FS 026 Immunology, Serology and Transplantation
004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
005 General Pathology and Pathological Anatomy
LA French
SL French; English
ED Entered STN: 7 Aug 1997
Last Updated on STN: 7 Aug 1997
AB The HIV infection paradox is that it induces a hyper-activation of the immune system resulting in an ***ineffective*** ***immune*** ***response*** . In this context, the cytokine network, which plays a key role in the regulation of cellular interactions within the immune system, is highly challenged. An imbalance in the cytokine profile resulting from cytokine disruption by HIV can be associated to disease progression, and cytokines may be involved in viral replication. Cytokines, cytokine inhibitors, and several cell derived anti-viral factors are proposed in immune based therapeutic strategies. In this review we attempt to analyse the involvement of cytokines in HIV-induced immune hyper-activation, the consequences of HIV infection on the cytokine imbalanced and T-cell defect and the appearance of HIV related haematological disorders and oncogenesis. Potential use of cytokine detection for prognostic purposes and insights in immune-based therapeutic strategies are discussed.

L6 ANSWER 53 OF 68 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 1996331440 EMBASE <<LOGINID::20100316>>
TI Immunology of Hodgkin's disease.
AU Poppema, S., Prof. (correspondence)
CS Department of Pathology, University of Groningen, University Hospital, Oostersingel 63, 9713 EZ Groningen, Netherlands.
SO Bailliere's Clinical Haematology, (1996) Vol. 9, No. 3, pp. 447-457.
ISSN: 0950-3536 CODEN: BCHAEW

CY United Kingdom
DT Journal; General Review; (Review)
FS 016 Cancer
025 Hematology
026 Immunology, Serology and Transplantation
005 General Pathology and Pathological Anatomy
LA English
SL English
ED Entered STN: 25 Nov 1996
Last Updated on STN: 25 Nov 1996
AB Hodgkin's disease is characterized by an immune response in the involved tissues that is predominantly CD4 mediated. The CD4+ T-cells are CD45RO+ and CD45RBdim, they express several activation markers but lack CD26, and in vitro can be stimulated to produce .gamma.-interferon and IL-4, but not IL-2. This is not the usual immunophenotype and cytokine production pattern of Th1, Th2 or Th0 cells and may be a reflection of anergy. The cause of such an anergic reaction is not clear since RS cells express HLA class II as well as the co-stimulator molecules CD80 and CD86. It is possible that a (hypothetical) super antigen expressed on the RS cells may play a role. The absence of IL-2 production however explains the absence of a CD8 mediated response. In addition to that, RS cells generally do not express HLA class I, which allows them to escape CD8 mediated responses. The link between the ***ineffective*** ***immune*** ***response*** in the tissue and the generalized immune deficiency in Hodgkin's disease may consist. of several components. These include the influx of mature T-cells into the affected tissues, the secretion of inhibitory molecules by the neoplastic cells and the spill-over of the anergic T-cell response into the general circulation by either the Hodgkin related antigen or also as a result of an IL-4 dominated response. The latter possibility may also be related to the hyper-gamma-globulinaemia and the frequently observed high IgE levels.

L6 ANSWER 54 OF 68 EMBASE
AN 1989010150 EMBASE <<LOGINID::20100316>>
TI Prevention of OPSI: Antibiotics and vaccines.
AU Perry Jr., J.F.
CS Division of Surgery, University of Minnesota Medical School, St. Paul, MN, United States.
SO Current Problems in Surgery, (1988) Vol. 25, No. 12, pp. 805-810.
ISSN: 0011-3840 CODEN: CPSUA7
CY United States
DT Journal
FS 025 Hematology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
007 Pediatrics and Pediatric Surgery
009 Surgery
LA English
SL English
ED Entered STN: 12 Dec 1991
Last Updated on STN: 12 Dec 1991
AB Prophylactic antibiotics appear to be effective in prevention of OPSI but remain unproven. Because the complication is not common, convincing patients to continue long-term penicillin prophylaxis is difficult, and noncompliance increases with time. Because of the ***ineffective***

immune ***response*** to vaccination with pneumococcal vaccine, young asplenic children should be given prophylactic antibiotics, preferably penicillin. This is especially true for the child 2 years of age or younger (in whom pneumococcal vaccine is not recommended). Any asplenic subject who develops a febrile illness should receive high-dose, broad spectrum antibiotics to which the pneumomoccus and other encapsulated bacteria are sensitive with no delay. When pneumococcal vaccines are given to asplenic persons, there may not be adequate antibody response to some pneumococcal polysaccharide serotypes. This may be especially manifest in younger children. Pneumococcal infections can also occur secondary to types of pneumococci not included in the vaccine. The vaccine is therefore, at best, only partially effective in prevention of OPSI. It remains the best available preventive measure against OPSI in the long term. The admonition to promptly treat any febrile illness with broad-spectrum antibiotics to which common infecting encapsulated bacteria are sensitive is equally applicable to the asplenic person who has received pneumococcal vaccine as to the very young child.

L6 ANSWER 55 OF 68 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 1986225480 EMBASE <<LOGINID::20100316>>
TI Immunoglobulin in chronic inflammatory disease.
AU Cottier, H.; Hassig, A.
CS Institute of Pathology, University of Bern, CH-3007 Bern, Switzerland.
SO Vox Sanguinis, (1986) Vol. 51, No. SUPPL. 2, pp. 39-43.
ISSN: 0042-9007 CODEN: VOSAAD
CY Switzerland
DT Journal; Article
FS 048 Gastroenterology
037 Drug Literature Index
031 Arthritis and Rheumatism
026 Immunology, Serology and Transplantation
025 Hematology
013 Dermatology and Venereology
012 Ophthalmology
LA English
ED Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991
AB Several case studies are presented to illustrate the success of intravenous gammaglobulin (IVIG) therapy in ulcerative colitis, Crohn's disease, chronic anterior uveitis, herpes zoster, and relapsing genital herpes simplex. Hypothetical mechanisms explain the marked inprovement of the chronic disorders, with particular emphasis on substitution of specific antibodies. The recently recognized heterogeneity of the major histocompatibility complex (MHC) is explored as one possible explanation for low and high responders to particular antigens. It is suggested that various chronic inflammatory disorders result from ***ineffective*** ***immune*** ***responses*** , and that the administration of IVIG may shift the delicate balance between the pathogen and the host to favor the latter.

L6 ANSWER 56 OF 68 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 1986180063 EMBASE <<LOGINID::20100316>>
TI Intravenous immunoglobulins: Pharmacological aspects and therapeutic use.
AU Hassig, A.
CS Central Laboratory, Swiss Red Cross Blood Transfusion Service, CH-3000

Bern 22, Switzerland.
SO Vox Sanguinis, (1986) Vol. 51, No. 1, pp. 10-17.
ISSN: 0042-9007 CODEN: VOSAAD
CY Switzerland
DT Journal; General Review; (Review)
FS 025 Hematology
026 Immunology, Serology and Transplantation
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LA English
ED Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991
AB The requirements for a present-day IVIG preparation are outlined. These are mainly: fully preserved activities of the recognition and effector functions of the IgG molecule, a normal subclass distribution, and a normal half-life after infusion. The therapeutic uses of IVIG preparations are discussed as follows: (1) Antibody substitution in cases of generalized or partial antibody deficiency in immune-compromised patients. These include the following diseases: hypogammaglobulinemia (congenital and acquired, including the neonates); drug-induced and viral immunosuppression. (2) Antibody stimulation in cases of selective antibody deficiency in otherwise immune-competent patients. These include acute cases of consumptive antibody deficiencies of the Jarisch-Herxheimer reaction type; in particular, chronic inflammations which apparently involve ***ineffective*** ***immune*** ***responses*** in which the organism is unable to build up sufficient amounts of antibodies with the required partial specificity, which is indispensable for overcoming the disease. (3) Modulation of the immune system by Ig-Ig interactions (mainly idiotype-anti-idiotype interactions) and Ig-Fc-receptor interactions, as it is known from the RES blockade during IVIG treatment of idiopathic thrombocytopenic purpura.

L6 ANSWER 57 OF 68 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 1985024985 EMBASE <<LOGINID::20100316>>
TI The importance of cytotoxic cellular immunity in the protection from cytomegalovirus infection.
AU Quinnan Jr., G.V.; Rook, A.H.
CS The Division of Virology, Office of Biologics, National Center for Drugs and Biologics, Food and Drug Administration, Bethesda, MD 20205, United States.
SO Birth Defects: Original Article Series, (1984) Vol. 20, No. 1, pp. 245-261.
ISSN: 0547-6844 CODEN: BTHDAK
CY United States
DT Journal; General Review; (Review)
FS 022 Human Genetics
026 Immunology, Serology and Transplantation
037 Drug Literature Index
007 Pediatrics and Pediatric Surgery
LA English
ED Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991
AB In these studies the authors have confirmed the widely held clinical impression that CMI is much more important than humoral immunity in determining the outcome of infection. In anticipation of this finding, the authors have designed studies to determine which defect in effector

cells correlate with a poor outcome of infection, and then to study the specific defects responsible for ***ineffective*** ***immune*** ***responses*** . The principal immune functions that have emerged as important correlates with outcome of infection are various types of HLA-restricted CTLs and nonrestricted non-T lymphocytes with characteristics of NK cells or antibody-dependent killer cells. This review summarizes the general characteristics of these responses, the evidence that they are important in determining the outcome of CMV infection, and the immune processes required for their development. In regard to the latter aspect, the approach used to define the reasons for defective cytotoxic responses when they occur is reviewed.

L6 ANSWER 58 OF 68 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 1978312683 EMBASE <<LOGINID::20100316>>
TI In vivo anti-tumor immunity detected by leukocyte adherence inhibition.
AU Gipson, T.G.; Martin, W.J.
CS Div. Virol., Bur. Biol., FDA, Bethesda, Md. 20014, United States.
SO Cancer Immunology, Immunotherapy, (1978) Vol. 3, No. 3, pp. 201-205.
ISSN: 0340-7004 CODEN: CIIMDN
CY Germany
DT Journal
FS 016 Cancer
025 Hematology
026 Immunology, Serology and Transplantation
LA English
AB The immune response of mice to a transplacentally induced alveolar cell tumor was studied with the leukocyte adherence inhibition (LAI) assay. The lung tumor, designated 85, was induced in a C3HfB/HeN (C3Hf) mouse by 1-ethyl-1-nitrosourea (ENU). While a dose of 105 cells of this tumor does not grow in syngeneic C3Hf mice, it does grow readily in (A x C3Hf)F1 hybrid mice. The tumor possesses a tumor associated transplantation antigen (TATA) which cross-reacts with a normal tissue alloantigen in strain A/HeN (A) mice. Normal mice, tumor-immunized C3Hf mice, and tumor-bearing (A x C3Hf)F1 mice possessed peritoneal cells, the majority of which adhered rapidly to glass and resisted gentle washing. When incubated with an extract of the 85 tumor, peritoneal cells from tumor-immunized mice demonstrated marked inhibition of adherence (62.4%) compared to similarly incubated peritoneal cells of either normal mice (30.3%) or tumor bearing mice (37.1%). Specificity of the reactivity in the LAI assay was demonstrated with a neuroblastoma extract and peritoneal cells from neuroblastoma-immunized C3Hf mice. Peritoneal cells from lung tumor-immunized mice, but not tumor-bearing mice, responded to a lung extract from strain A mice. In contrast to the microcytotoxicity assay, the LAI assay is capable of distinguishing the effective anti-tumor response of tumor-immunized C3Hf mice from the ***ineffective*** ***immune*** ***response*** of tumor-bearing (A x C3Hf)F1 mice.

L6 ANSWER 59 OF 68 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 1975162156 EMBASE <<LOGINID::20100316>>
TI Hepatitis B antigenemia, specific immune deficiency and hepatocellular carcinoma.
AU Simons, M.J.; Yu, M.; Shanmugaratnam, K.
CS WHO Immunol. Res. Train. Cent., Univ. Singapore, Singapore.
SO Tumor Research, (1973) Vol. Vol. 8, pp. 120-126.
ISSN: 0041-4093 CODEN: TUREA6

DT Journal
FS 016 Cancer
026 Immunology, Serology and Transplantation
048 Gastroenterology
006 Internal Medicine
LA English
AB One of the major objectives of studies involving Hepatitis B antigen (HB Ag) is to determine whether HB antigenemia following exposure to the HB agent is a risk factor in the development of hepatocellular carcinoma (HCC). An increased frequency of HB Ag was demonstrated in the sera of patients with HCC and those with other chronic liver diseases. It has been claimed that these findings provide circumstantial evidence in support of the view that persistent HB antigenemia (the 'carrier state') predisposes to the development of HCC. There appear to be 2 main modes of immune responsiveness on exposure to the HB agent; development either of immunity with the production of antibody to HB Ag in the absence of detectable antigen, or of HB antigenemia with or without specific antibody (HB Ab). In the former situation, immune mechanisms appear to be effective in eliminating the HB agent. By contrast, the carrier state of HB antigenemia probably reflects an ***ineffective*** ***immune*** ***response*** and consequent persisting infection. Based on the foregoing, it is postulated that exposure to the HB agent, a precondition for the development of HB antigenemia may cause HCC either directly or indirectly through one of the types of post hepatitic pathology. Evidence relating to this proposition may be obtained by answering the following questions. Do HCC patients differ from suitable ethnic, sex and age matched normal subjects and patients with a variety of liver diseases in HB agent exposure rate, or in the proportion of antigenemic persons in the exposed population (HB antigenemic rate)? Do individuals who are infected by the HB agent, and in particular those who become HB Ag 'carriers', develop HCC more frequently than individuals who are not exposed? Could the incidence of HCC be decreased by the introduction of measures directed towards reducing the risk of exposure and in particular of developing HB antigenemia (eg. vaccination of the population at risk, elimination of the HB agent)? The present status of some studies in progress in Singapore relating to the first of these questions is summarised in this paper.

L6 ANSWER 60 OF 68 MEDLINE on STN
AN 2010103970 IN-PROCESS <<LOGINID::20100316>>
DN PubMed ID: 19828908
TI Acute enterovirus infection followed by myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS) and viral persistence.
AU Chia J; Chia A; Voeller M; Lee T; Chang R
CS EV Med Research, Torrance, California, USA.. evmed@sbcglobal.net
SO Journal of clinical pathology, (2010 Feb) Vol. 63, No. 2, pp. 165-8.
Electronic Publication: 2009-10-14.
Journal code: 0376601. E-ISSN: 1472-4146. L-ISSN: 0021-9746.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS NONMEDLINE; IN-PROCESS; NONINDEXED; Abridged Index Medicus Journals; Priority Journals
ED Entered STN: 16 Feb 2010
Last Updated on STN: 27 Feb 2010
AB AIMS: Enteroviruses are well-known causes of acute respiratory and/or gastrointestinal infections and non-specific flu-like illness. Although enterovirus protein, RNA and non-cytopathic viruses have been demonstrated

in the stomach biopsies of patients with myalgia encephalomyelitis/chronic fatigue syndrome (ME/CFS), causality for chronic diseases is difficult to establish without having well-documented cases of acute enterovirus infections. The aim of this study was to link acute enteroviral infection to viral persistence in patients with ME/CFS. METHOD: Patients admitted to the hospital with acute febrile illnesses were screened for enteroviral infections. Acutely infected patients were followed longitudinally, and those who developed symptoms of ME/CFS underwent oesophagogastroduodenoscopy and biopsies of the antrum to document viral persistence by immunoperoxidase staining for viral protein and viral RNA assay. RESULTS: Three representative patients with different manifestations of acute enterovirus infections progressed to have chronic symptoms of ME/CFS. Persistent viral infection was demonstrated in the antrum years later. CONCLUSION: After acute infections, enteroviruses can persist in patients resulting in manifestation of ME/CFS. Chronic enterovirus infection in an immunocompetent host may be an example of a stalemate between attenuated, intracellular viruses and an ***ineffective*** ***immune*** ***response*** .

L6 ANSWER 61 OF 68 MEDLINE on STN
AN 2009825640 MEDLINE <<LOGINID::20100316>>
DN PubMed ID: 20010143
TI Hemophagocytic lymphohistiocytosis in the premature neonate: a case study.
AU Woods Amanda G; Woods Christopher W
CS Women's Hospital of Greensboro, Greensboro, North Carolina 27408, USA.. woodsland@yahoo.com
SO Advances in neonatal care : official journal of the National Association of Neonatal Nurses, (2009 Dec) Vol. 9, No. 6, pp. 274-8.
Journal code: 101125644. E-ISSN: 1536-0911. L-ISSN: 1536-0903.
CY United States
DT (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 201003
ED Entered STN: 22 Dec 2009
Last Updated on STN: 5 Mar 2010
Entered Medline: 4 Mar 2010
AB Hemophagocytic lymphohistiocytosis (HLH) is a rare disease resulting from an abnormal proliferation of histiocytes within the body's tissues leading to an ***ineffective*** ***immune*** ***response*** . Typically, HLH is characterized by fever, hepatosplenomegaly, cytopenia, hypertriglyceridemia, hypofibrinogenemia, and hemophagocytosis. However, the premature infant with HLH may present differently making diagnosis of the disease cumbersome. If an infant is born with ascites, cytopenias, hypofibrinogenemia, and hepatosplenomegaly, a diagnosis of HLH cannot be ruled out. In addition, premature infants oftentimes will not present with fever because they are kept normothermic from ambient sources. Reports of premature infants with HLH in the literature are rare. This is a case presentation of a 27-week-gestation female with a family history of HLH.

L6 ANSWER 62 OF 68 MEDLINE on STN
AN 2009580638 MEDLINE <<LOGINID::20100316>>
DN PubMed ID: 19707989
TI Hemophagocytic lymphohistiocytosis: when the immune system runs amok.
AU Janka G

CS Pediatric Hematology and Oncology, University Medical Center Hamburg-Eppendorf, Hamburg, Germany.. janka@uke.uni-hamburg.de
SO Klinische Padiatrie, (2009 Sep) Vol. 221, No. 5, pp. 278-85. Electronic Publication: 2009-08-25. Ref: 71
Journal code: 0326144. E-ISSN: 1439-3824. L-ISSN: 0300-8630.
CY Germany: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 200911
ED Entered STN: 27 Aug 2009
Last Updated on STN: 16 Dec 2009
Entered Medline: 30 Nov 2009
AB Hemophagocytic lymphohistiocytosis (HLH) is a life-threatening syndrome in which an exaggerated but ***ineffective*** ***immune*** ***response*** leads to severe hyperinflammation. Key players in HLH are activated lymphocytes and histiocytes which infiltrate all organs and secrete large amounts of cytokines. Cardinal symptoms are prolonged fever, hepatosplenomegaly, cytopenias, and hemophagocytosis. Biochemical markers include elevated ferritin, triglycerides, and low fibrinogen. HLH occurs on the basis of various inherited and acquired immune defects. Impaired function of natural killer cells and cytotoxic T cells is shared by all forms of HLH. Nearly all genetic defects identified in inherited cases of HLH are either mutations in the perforin gene or in genes important for the exocytosis of cytotoxic granules. Cytotoxic granules contain perforin and granzymes which induce apoptosis upon entering the target cell. Additionally perforin is important for the down-regulation of the immune response. Acquired forms of HLH are found in association with infectious agents, in patients with autoimmune diseases, in malignant diseases, and in patients receiving immune suppression or after organ transplantation. - HLH is still difficult to diagnose and may be overlooked since initially it may masquerade as a normal infection. HLH should be considered when symptoms are more pronounced than usual and in case of progression. Suppression of the severe hyperinflammation can be achieved with immunosuppressive and immunomodulatory agents and cytostatic drugs. Patients with genetic HLH have to undergo stem cell transplantation for cure.

L6 ANSWER 63 OF 68 MEDLINE on STN
AN 2009228716 MEDLINE <<LOGINID::20100316>>
DN PubMed ID: 19308259
TI Necator americanus infection: a possible cause of altered dendritic cell differentiation and eosinophil profile in chronically infected individuals.
AU Fujiwara Ricardo T; Cancado Guilherme G L; Freitas Paula A; Santiago Helton C; Massara Cristiano Lara; Dos Santos Carvalho Omar; Correa-Oliveira Rodrigo; Geiger Stefan M; Bethony Jeffrey
CS Laboratory of Cellular and Molecular Immunology, Instituto Rene Rachou, Oswaldo Cruz Foundation, Belo Horizonte, Minas Gerais, Brazil.
SO PLoS neglected tropical diseases, (2009) Vol. 3, No. 3, pp. e399. Electronic Publication: 2009-03-24.
Journal code: 101291488. E-ISSN: 1935-2735. L-ISSN: 1935-2727.
Report No.: NLM-PMC2654967.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English
FS Priority Journals
EM 201003
ED Entered STN: 25 Mar 2009
Last Updated on STN: 9 Mar 2010
Entered Medline: 8 Mar 2010
AB BACKGROUND: Hookworms survive for several years (5 to 7 years) in the host lumen, inducing a robust but largely ***ineffective*** ***immune*** ***response*** . Among the most striking aspects of the immune response to hookworm (as with many other helminths) is the ablation of parasite-specific T cell proliferative response (hyporesponsiveness). While the role of the adaptive immune response in human helminth infection has been well investigated, the role of the innate immune responses (e.g., dendritic cells and eosinophils) has received less attention and remains to be clearly elucidated. METHODOLOGY/PRINCIPAL FINDINGS: We report on the differentiation/maturation of host dendritic cells in vitro and the eosinophil activation/function associated with human hookworm infection. Mature DCs (mDCs) from Necator americanus (Necator)-infected individuals showed an impaired differentiation process compared to the mDCs of non-infected individuals, as evidenced by the differential expression of CD11c and CD14. These same hookworm-infected individuals also presented significantly down-regulated expression of CD86, CD1a, HLA-ABC, and HLA-DR. The lower expression of co-stimulatory and antigen presentation molecules by hookworm-infected-derived mDCs was further evidenced by their reduced ability to induce cell proliferation. We also showed that this alternative DC differentiation is partially induced by excreted-secreted hookworm products. Conversely, eosinophils from the same individuals showed a highly activated status, with an upregulation of major cell surface markers. Antigen-pulsed eosinophils from N. americanus-infected individuals induced significant cell proliferation of autologous PBMCs, when compared to non-infected individuals. CONCLUSION: Chronic N. americanus infection alters the host's innate immune response, resulting in a possible modulation of the maturation process of DCs, a functional change that may diminish their ability for antigen presentation and thus contribute to the ablation of the parasite-specific T cell proliferative response. Interestingly, a concomitant upregulation of the major cell surface markers of eosinophils was observed in hookworm-infected individuals, indicative of antigen-specific immune responses, especially antigen presentation. We showed that in addition to the postulated role of the eosinophils as effector cells against helminth infection, activated cells may also be recruited to sites of inflammation and contribute to the immune response acting as antigen presenting cells.

L6 ANSWER 64 OF 68 MEDLINE on STN
AN 2000226983 MEDLINE <<LOGINID::20100316>>
DN PubMed ID: 10765916
TI Report on the Fourth International Workshop on Reactive Arthritis.
AU Sieper J; Braun J; Kingsley G H
CS Department of Medicine, University Hospital Benjamin Franklin, and German Rheumatology Research Center, Berlin.
SO Arthritis and rheumatism, (2000 Apr) Vol. 43, No. 4, pp. 720-34.
Journal code: 0370605. ISSN: 0004-3591. L-ISSN: 0004-3591.
CY United States
DT Conference; Conference Article; (CONGRESSES)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Abridged Index Medicus Journals; Priority Journals

EM 200005
ED Entered STN: 25 May 2000
Last Updated on STN: 25 May 2000
Entered Medline: 18 May 2000
AB There are large differences in the antigenicity and biology of the ReA-associated bacteria. For induction of arthritis, the relevance seems to be only that antigenic material reaches the joint, alive or dead. If there is a common antigen, it has to be a highly conserved one. Bacterial hsp60 seems to be an immunodominant T cell antigen in ReA, but there must be other relevant antigens shared by these different bacteria. An ***ineffective*** ***immune*** ***response*** (for example, low production of TNFalpha) seems to contribute to the manifestations and course of ReA. Although arthritis can also occur in its absence, HLA-B27 plays an important role in the pathogenesis of ReA and the other SpA. Current data suggest that B27 probably acts as an antigen-presenting molecule for a still-unknown arthritogenic molecule. Comparison of ReA with IBD-associated arthritis suggests that there might indeed be a common antigen shared by ReA-associated bacteria and bacteria of the gut flora. CD8+ T cells seem to be important in ReA and other SpA. In some parts of the world, such as in Mexico, ReA could be a major predisposing cause of the development of AS. Antibiotic treatment is not effective, probably because the triggering bacteria are already dead or in a partly latent state at the time arthritis occurs. Based on this knowledge and on new technologies, it should be possible in future years to derive answers to the questions about ReA and the other SpA and, as a consequence, to find a cure.

L6 ANSWER 65 OF 68 MEDLINE on STN
AN 1996291447 MEDLINE <<LOGINID::20100316>>
DN PubMed ID: 8691051
TI Idiopathic thrombocytopenic purpura (ITP): immunomodulation by intravenous immunoglobulin (IVIg).
AU Imbach P; Morell A
CS Inselspital, Bern, Switzerland.
SO International reviews of immunology, (1989) Vol. 5, No. 2, pp. 181-8.
Ref: 34
Journal code: 8712260. ISSN: 0883-0185. L-ISSN: 0883-0185.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 199608
ED Entered STN: 11 Sep 1996
Last Updated on STN: 11 Sep 1996
Entered Medline: 29 Aug 1996
AB ITP is a destructive thrombocytopenia. Platelets are coated with antibodies and these opsonized platelets are rapidly removed by phagocytes. On the other hand, therapeutic application of antibody concentrates (IVIg) rapidly raise platelet counts, and in some patients, sustained platelet recovery has been observed. The mechanism of action of IVIg is far from being clear. Several possible mechanism of action of IVIg treatment have been described. The immediate effect of IVIg seems to be a decrease in (unspecific) Fc mediated mononuclear phagocytosis, the long term effect might be a change in the complex network of the regulatory function of the immune response. Both types of interactions seem to play a keyrole in the immunomodulation. The various possible

modes of actions evoke investigation of IVIg in a wide range of diseases with similar ***ineffective*** ***immune*** ***response*** . Controlled clinical studies have to be done to prove or disapprove the use of IVIg in other indications.

L6 ANSWER 66 OF 68 MEDLINE on STN
AN 1996170456 MEDLINE <<LOGINID::20100316>>
DN PubMed ID: 8600684
TI [Immunopathology of chronic liver diseases].
Immunpathologie chronischer Lebererkrankungen.
AU Meyer zum Buschenfelde K H
CS I. Medizinische Klinik und Poliklinik, Johannes Gutenberg-Universitat Mainz.
SO Verhandlungen der Deutschen Gesellschaft fur Pathologie, (1995) Vol. 79, pp. 186-97. Ref: 38
Journal code: 7503704. ISSN: 0070-4113. L-ISSN: 0070-4113.
CY GERMANY: Germany, Federal Republic of
DT (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA German
FS Priority Journals
EM 199604
ED Entered STN: 13 May 1996
Last Updated on STN: 13 May 1996
Entered Medline: 26 Apr 1996
AB Chronic inflammatory liver diseases can be induced by virus infections, toxic-metabolic factors and/or autoimmune mechanisms. This overview deals with the immunopathogenesis of chronic hepatitis B and C and autoimmune hepatitis (AIH). 1. Chronic hepatitis B: The immune response to HBV-antigens is responsible both for viral clearance and disease pathogenesis during HBV-infection. The humoral immune response to HBsAg contributes to the clearance of circulating virus particles, the cell mediated immune response to HBsAg, HBcAg and polymerase antigen eliminates infected cells. The class I- and class II restricted T-cell-responses to HBV is strong, polyclonal and multispecific in acute HB with successful clearance of the virus, but weak or incomplete in chronic HB with viral persistence. In addition to ***ineffective*** ***immune*** ***response*** host and viral factors as well as abnormalities in virus-host interactions may be the main reasons for the maintenance of HBV-carrier status. 2. Chronic hepatitis C develop in more than 60% of infected patients. There is increasing evidence that the immune response to HCV-epitopes plays an important role in the course and the pathogenesis of the disease. It has been shown that CD4+ and CD8+ T-cells recognize viral peptides in the presence of class I and II molecules. The fine specificity and functional significance of liver infiltrating and peripheral blood T-cells demonstrate HCV specific immunodominant epitopes targeted by class Ii restricted CD4+ cells in patients with chronic HCV infection. The T-cell response correlates with disease activity. The cytokine release of T-cells resemble a TH1-like profile. Studies of the humoral immune response to HCV show a correlation between IgM-anti-HCV and disease activity. In vitro and in vivo anti-HCV secretion by PBMC is due to persistent antigenic stimulation of B-cells by ongoing production of viral antigens and reflects HCV replication in PBMC. Of special interest are several immune mediated disease and immune abnormalities in chronic hepatitis C. 3. Autoimmune hepatitis (AIH) is a distinct group of acute and chronic necro-inflammatory disorders of unknown etiology characterized

by immunological and autoimmunological features including the presence of autoantibodies but without an antecedent of viral infections. Marker autoantibodies define 3 subtypes: Type I (ANA/SMA), Type II (LKM1-AB), Type II (SLA-AB). AIH is associated with a distinct genetic background (HLA A1, B8, DR3 or DR4). Several studies clearly demonstrate that liver cell damage in AIH is mediated by autoimmune reactions against normal constituents of hepatocytes. Although the precise mechanisms are not yet fully understood, there is now considerable evidence that autoantigens of the hepatocellular membrane in particular the ASGPR are important targets of liver damaging autoreactions in AIH. Cellular and humoral immune reactions against the human ASGPR correlate with disease activity and usually disappear under immunosuppressive therapy.

L6 ANSWER 67 OF 68 MEDLINE on STN
AN 1991345954 MEDLINE <<LOGINID::20100316>>
DN PubMed ID: 1878260
TI Neonatal tetanus despite protective serum antitoxin concentration.
AU Maselle S Y; Matre R; Mbise R; Hofstad T
CS Department of Microbiology and Immunology, Muhimbili Medical Centre, Tanzania.
SO FEMS microbiology immunology, (1991 Jun) Vol. 3, No. 3, pp. 171-5.
Journal code: 8901230. ISSN: 0920-8534. L-ISSN: 0920-8534.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 199110
ED Entered STN: 20 Oct 1991
Last Updated on STN: 3 Feb 1997
Entered Medline: 3 Oct 1991
AB Using the ELISA technique to estimate serum antibodies against tetanus toxin, seven neonates with clinical tetanus were found to have antibody levels 4-13 times higher than the presumed minimum protective level of 0.01 IU/ml. All but one of their mothers had been vaccinated with tetanus toxoid in pregnancy. In two other neonates, whose mothers had received multiple booster doses of toxoid during pregnancy, the anti-toxin concentrations were 100- and 400-times the presumed protective level. Therefore the toxin dose may overwhelm the pre-existing anti-toxin level and produce disease. Furthermore, multiple booster injections of tetanus toxoid may not only enhance serum anti-toxin titres, but could also lead to an ***ineffective*** ***immune*** ***response*** .

L6 ANSWER 68 OF 68 MEDLINE on STN
AN 1975212222 MEDLINE <<LOGINID::20100316>>
DN PubMed ID: 50354
TI Normal tissue alloantigens and genetic control of susceptibility to tumors: microcytotoxicity studies on resistant C3Hf and susceptible (A X C3Hf) F1 mice inoculated with transplacentally induced C3Hf lung tumor.
AU Martin W J; Esber E; Cotton W G; Rice J M
SO Journal of immunology (Baltimore, Md. : 1950), (1975 Jul) Vol. 115, No. 1, pp. 289-295.
Journal code: 2985117R. ISSN: 0022-1767. L-ISSN: 0022-1767.
CY United States
DT (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
LA English

FS Abridged Index Medicus Journals; Priority Journals
EM 197511
ED Entered STN: 10 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 6 Nov 1975
AB The immune response of mice to a transplacentally induced lung tumor was investigated with the microcytotoxicity (MC) assay. The tumor, originally induced in C3Hf mice, does not grow readily when transplanted to normal syngeneic C3Hf recipients. It grows readily, however, in (A C3Hf)F1 hybrids and in strain C3H mice, which express in their normal lung tissue a component which constitutes a strong lung tumor-associated transplantation antigen (TATA) in C3Hf mice. Both lung tumor-immunized C3Hf and tumor-bearing (A X C3Hf)F1 and C3H mice possessed lymphoid cells reactive against cultured lung tumor cells in the MC assay. Reactivity was also observed against cells cultured from normal lungs of (A X C3Hf)F1 and C3H mice, but not against cells similarly cultured from C3Hf of C57BL/6 mice. Anti-tumor MC was inhibited by serum-blocking factors present in some but not all tumor-bearing and tumor-immunized mice. The MC assay and detection by it of serum-blocking factors does not distinguish the effective anti-C3Hf lung tumor immune response of immunized C3Hf mice from the ***ineffective*** ***immune*** ***response*** of tumor-bearing (A X C3Hf)F1 and C3H mice. Furthermore, in lung tumor-bearing mice cells reactive in the MC assay may be directed against a normal tissue antigen rather than a tumor-associated antigen.